LIBRARY
BOSTON UNIVERSITY
SCHOOL OF MEDICINE

The Journal of
Thoracic Surgery

VOL. 12 OCTOBER, 1942 No. 1

Original Communications

FURTHER EXPERIENCES IN THE SURGICAL TREATMENT OF SUBACUTE STREPTOCOCCUS VIRIDANS ENDARTERITIS SUPERIMPOSED ON PATENT DUCTUS ARTERIOSUS

ARTHUR S. W. TOUROFF, M.D., F.A.C.S.
NEW YORK, N. Y.

THE first report of an attempt at surgical cure of subacute bacterial endarteritis complicating patent ductus arteriosus was made by Graybiel, Strieder, and Boyer[5] in 1938. The case, which incidentally also represented the very first effort at ductal ligation to be recorded in the literature, unfortunately terminated fatally on the fourth postoperative day. (The first successful ligation of a *noninfected* patent ductus arteriosus was reported by Gross and Hubbard[9] in 1939.)

The first case of patent ductus arteriosus complicated by subacute bacterial endarteritis, in which operation was followed by recovery from infection, was presented by Touroff and Vesell[18] before the Twenty-Third Annual Meeting of The American Association for Thoracic Surgery in June, 1940.* The case was one of a group of four which had been operated upon, by the present author, in the hope of curing superimposed subacute *Streptococcus viridans* endarteritis. The results in these four operative cases,[17] which were the first to be presented following the unsuccessful case of Graybiel, Strieder and Boyer, were as follows: In the first, recovery ensued. (The patient remains entirely well, more than two years following operation.) In the second case, operation had only transient beneficial effect because vegetations apparently already were present upon the aortic valve. (This patient succumbed of the original subacute *Streptococcus viridans* infection, eight months later.) In the two remaining cases death, from uncontrollable hemorrhage, occurred in the operating room.

From Beth Israel Hospital and The Mount Sinai Hospital.
Received for publication March 14, 1942.

*In July, 1940, Keele and Tubbs[11] of London reported a case of infected patent ductus in which, following operation, the patient survived but was not cured of infection.

Since subacute *Streptococcus viridans* endarteritis previously had been considered an almost invariably fatal disease, the prompt recovery of one patient of the two who survived operation was heartening. However the death of the third and fourth patients, from accidental hemorrhage, tended to indicatet hat operation in the presence of infected patent ductus was attended with considerable risk. Accordingly, the viewpoint was expressed that surgical therapy appeared worthy of further trial but that the development of some modification of operative technique, to reduce the hazards of operation, was highly desirable. In view of the fact that the recovery of the first patient might have been entirely fortuitous, it was emphasized that an evaluation of the effectiveness of surgical therapy would have to await further experience.

During the ensuing year, a modification of the operative technique of Gross[6] was devised by the author;[12] and since then four additional patients, suffering from relatively early infection, have been subjected to operation.* The purpose of this communication is to present these cases briefly and to discuss certain aspects of infected patent ductus arteriosus which bear a relationship to surgical therapy.

CASE REPORTS

CASE 5.—F. S., female, aged 29 years.[19] Patent ductus arteriosus was discovered at the age of 8 years. She was admitted to Beth Israel Hospital on May 15, 1941, with a suspected fracture of the left hip. Fever was present on admission. Roentgenologic examination of the hip was negative. The temperature remained elevated without obvious cause. Blood culture, performed several days after admission, contained *Streptococcus viridans* (fifteen colonies per cubic centimeter). The patient was observed in the hospital for thirty-one days. The course was progressively downhill. The temperature ranged between 98.6° and 104° F. Additional blood cultures contained as many as 177 colonies of *Streptococcus viridans* per cubic centimeter. Chemotherapy was poorly tolerated. Blood culture taken on the *morning of operation* contained *80 colonies per cubic centimeter.*

Operation (June 14, 1941) consisted of double ductal ligation. The ductus was indurated and moderately adherent. The *first blood culture,* taken *three hours* postoperatively, was sterile. *Eleven* blood cultures were performed subsequently, and *these also were sterile.*

Sulfapyridine was administered on the twelfth postoperative day, because of continuing fever without obvious cause. On the following morning, a small abscess of the superficial wound was discovered and evacuated. Chemotherapy was discontinued at once, the patient having received only 10 Gm. of the drug in all. *All* postoperative blood cultures—six in number—performed *prior* to the administration of sulfapyridine, were *negative. Chemotherapy therefore cannot be credited with contributing to the recovery.*

*Two patients with suspected patent ductus arteriosus, complicated by subacute *Streptococcus viridans* infection of *long duration,* also were operated upon. Both presented the stigmata of chronic bacteremia, i.e., profound anemia, café au lait color, great splenic enlargement, petechiae, amyloidosis, renal involvement, etc. (In one, a patent ductus was not found at operation and the nature of the existing, congenital cardiac anomaly still remains obscure.) In advance of operation, these two cases deliberately were placed in a special experimental group for the reason that complete recovery appeared to be most unlikely. They will not be described in this report, but will be the subject of a forthcoming, separate communication.

Convalescence was essentially uneventful thereafter. The patient was discharged from the hospital on the thirty-fourth postoperative day and has remained entirely well up to the present time.

CASE 6.—G. O., female, aged 51 years.[16] Patent ductus arteriosus was discovered during early adult life. In 1929, the patient had subacute bacterial endarteritis superimposed on the patent ductus. Numerous blood cultures were positive for *Streptococcus viridans* at the time. (There had been no history of rheumatic fever, previously.) Spontaneous recovery occurred after fifteen months.* Since 1934 the patient had been under medical supervision because of mild but progressive circulatory embarrassment.

The patient was admitted to The Mount Sinai Hospital, on Aug. 12, 1941, with a two and one-half weeks' history of fever, malaise, and weakness. A characteristic episode of left-sided pulmonary infarction had occurred five days prior to admission. Two blood cultures were taken in the hospital. These contained 40 and 52 colonies of *Streptococcus viridans* per cubic centimeter, respectively. The patient was observed in the hospital for twenty-three days. The course was progressively downhill. The temperature ranged between 98° and 103.6° F. Sulfapyridine and sulfathiazole were poorly tolerated, and symptoms of circulatory failure persisted.

Operation (Sept. 4, 1941) consisted of double ductal ligation. The ductus was only slightly indurated and adherent. Extensive intrapleural adhesions, the result of old and recent pulmonary infarctions, were present. The pulmonary artery was tremendously enlarged and covered the aortic arch completely.

The *first* postoperative blood culture, taken *forty minutes after actual ductal ligation,* was *sterile. Four* blood cultures were performed subsequently, and *these also were sterile.* Convalescence was uneventful. *Chemotherapy was not administered postoperatively.* The patient was discharged from the hospital thirteen days after operation and has remained entirely well up to the present time. Exercise tolerance is normal for the first time in many years.

CASE 7.—J. Z., female, aged 18 years.[14] Patent ductus arteriosus was discovered at the age of 1½ years. The patient was admitted to Beth Israel Hospital on Sept. 24, 1941, with a four-month history of dyspnea, lassitude, and high fever. A severe episode of left-sided pulmonary infarction had occurred two months after onset of illness. The patient had been confined to another hospital for the five and one-half weeks immediately preceding this admission. Blood cultures taken there were repeatedly positive for *Streptococcus viridans.* Sulfathiazole, in adequate dosage, was well tolerated but ineffectual. The clinical course was of the septic type, with temperature ranging as high as 105° F. Evidence of increasing circulatory failure developed.

On admission to Beth Israel Hospital, the patient was very acutely ill, septic, and greatly emaciated. A blood culture contained 70 colonies of *Streptococcus viridans* per cubic centimeter. The patient was observed for only three days. A preoperative blood culture, taken in the *operating room,* contained *120 colonies of organisms per cubic centimeter.*

Operation (Sept. 27, 1941) consisted of complete ductal division.† Extensive intrapleural adhesions were present. The ductus was markedly indurated and adherent. A fair-sized pulmonary infarct was noted in the left upper lobe. The mediastinal structures were markedly edematous, and many large, inflamed lymph

*This is the second spontaneous recovery from subacute bacterial endarteritis complicating patent ductus arteriosus, to be reported. The first such case was reported by Chester.[3]

†This ductal division and the one which was performed in our original case,[18] represent the only two in the literature up to the present time.

nodes were noted. Slight bleeding from the ductus occurred during the last stage of ductal dissection. The ductus was readily clamped, doubly ligated, and divided.

The *first* postoperative blood culture, taken in the operating room *eleven minutes after actual ductal division,* contained *one colony of Streptococcus viridans* per cubic centimeter. A second culture taken *thirty minutes later* also contained *one colony* per cubic centimeter. *Seven* blood cultures were performed subsequently, *and all were sterile.* Convalescence was uneventful. *Chemotherapy was not administered postoperatively.* The patient was discharged from the hospital twenty-six days after operation and has remained entirely well up to the present time.

CASE 8.—H. N., male, aged 9 years. Patent ductus arteriosus was discovered at the age of 8 months. The patient was admitted to The Mount Sinai Hospital Jan. 1, 1942, because of fever and malaise of twenty-four days' duration. He was confined to another hospital for nine days immediately preceding admission. While there, the temperature ranged between 103° and 104° F., and blood cultures contained 38 to 88 colonies of a gram-positive organism, identified as an "enterococcus." Sulfadiazine was well tolerated but ineffectual, except for some antipyretic effect. At The Mount Sinai Hospital, blood cultures on two occasions revealed *Streptococcus viridans* (2 and 8 colonies per cubic centimeter, respectively). The patient was observed in the hospital for eight days where sulfadiazine therapy was continued. A preoperative culture, taken in the *operating room,* contained *numerous colonies* of *Streptococcus viridans.*

Operation (Jan. 9, 1942) consisted of double ductal ligation. The ductus appeared entirely normal.

The *first* postoperative blood culture, taken in the operating room *ten minutes after ductal ligation,* was *sterile.* A *second* culture taken *twenty minutes later* also was *sterile.* A *third* culture, taken *one hour postoperatively,* likewise was *sterile.* A *fourth* culture, taken *two hours* postoperatively, contained *one colony* of *Streptococcus viridans* per *4 c.c. of blood.* A *fifth* culture, taken *seven hours* postoperatively, also contained *one colony* per *4 c.c. of blood.*

During the next six days, *four blood cultures* were taken, and *all were reported sterile.* On the eighth postoperative day, a culture revealed *1 colony in 20 c.c. of blood.* (This was the only one of the author's cases in which a positive culture was obtained after *negative* cultures once had become established.) *Six* blood cultures were taken subsequently at two-day intervals; *all were sterile.* Convalescence was uneventful. *Chemotherapy was not administered postoperatively.* The patient was discharged from the hospital twenty days after operation and has remained entirely well.

DISCUSSION OF OPERATIVE CASES

In the brief case reports presented above, there are included only those details which are germane to the present discussion. However, references to the more complete, individual reports will be found in the appended bibliography. In all four cases, the blood cultures were positive on admission to the hospital and chemotherapy was administered, or had been administered previously, in an attempt to bring about recovery before resorting to surgery. In two cases (Cases 5 and 6), sulfonamide drugs were poorly tolerated and had to be discontinued before adequate dosage could be established. In the remaining two (Cases 7 and 8), chemotherapy was well tolerated but proved ineffectual. Thus,

when referred for surgical treatment, all patients still presented evidence of active infection and had positive blood cultures. Operation consisted of ductal ligation in three instances and ductal division in one (Case 7).

In order to observe the effect of surgical therapy alone, it was planned not to employ chemotherapy after operation. However in one case (Case 5), sulfapyridine was administered on the *twelfth* postoperative day. This was discontinued on the following morning, the patient having received a total of only 10 Gm. of the drug in all. It is to be emphasized that in this case, *six* postoperative blood cultures had been taken *before the administration of chemotherapy was begun,* and *all had been reported sterile.* Accordingly, it is justifiable to state that the use of sulfapyridine played no role in the patient's recovery. In the remaining three cases, chemotherapy was not administered postoperatively. Under the circumstances, the recovery of *all* four patients can be attributed *solely to operation.* These results suggest most strongly that the recovery of our original patient was not fortuitous. Furthermore they permit the inference that operative cure now can be anticipated with some degree of regularity, at least in those cases in which the infection is of relatively short duration.

THE THEORETICAL DANGERS OF OPERATION

In our original presentation,[17] attention was called to the early opinion expressed by Gross,[7] namely, that operation was contraindicated in cases of infected patent ductus because "the friable vegetations will almost certainly be dislodged and result in embolism or a more severe bacteremia."* This viewpoint was not substantiated by observations made in the two patients who survived operation, in our first series. It is further negated by the four cases of the present series, for in not a single instance did *postoperative* embolization occur despite the fact that in two cases spontaneous pulmonary embolization was noted *prior* to operation. Postoperatively, more severe bacteremia not only did *not* occur, but the blood cultures became *sterile* in an astonishingly short period of time.

A question as to the likelihood of occurrence of postoperative *Streptococcus viridans* mediastinal and pleural infection also has been answered, for in none of the cases was such infection noted. Accordingly it appears warranted to state that, if the patient survives operation, there appears to be little danger of postoperative embolization, more severe bacteremia, or *Streptococcus viridans* mediastinal and pleural infection. Pyogenic infection from some extraneous source is, as in any surgical case, a possibility; and in one case (Case 5), an abscess of the superficial wound developed.

As was emphasized in our original presentation, the greatest danger of operation in cases of infected patent ductus is that of accidental

*As the result of recent personal experience Gross, in a communication to the author, has withdrawn his original objections to operation in the presence of infected patent ductus arteriosus.

TABLE I

SUBACUTE STREPTOCOCCUS VIRIDANS ENDARTERITIS SUPERIMPOSED ON PATENT DUCTUS ARTERIOSUS (FIVE CURED CASES)*

CASE NO.	PATIENT	AGE (YEARS)	DURATION OF INFECTION	PREOPERATIVE CHEMOTHERAPY	BLOOD CULTURES		OPERATION DATE AND TYPE	POSTOPERATIVE BLOOD CULTURES		POSTOPERATIVE CHEMOTHERAPY	RESULT
					NO. OF COLONIES PER C.C.	LAST PREOPERATIVE CULTURE		INITIAL	SUBSEQUENT		
1	S. S.	29	3 months	None	50-400	3 days preop., heavily positive	1/27/40 Ductal division	2 days postop. —sterile	6 All sterile	Sulfapyridine 9 grams, on 5th day (2 blood cultures already sterile)	Well
5	F. S.	29	31 (+) days (6-8 weeks) ?	Poorly tolerated	0-177	Day of operation, 80 colonies per c.c.	6/14/41 Ductal ligation	3 hours postop. —sterile	11 All sterile	Sulfapyridine 10 grams, on 12th day (6 blood cultures already sterile)	Well
6	G. O.	51	8 weeks	Poorly tolerated	40-52	10 days preop., 52 colonies per c.c.	9/4/41 Ductal ligation	40 minutes postop.— sterile	4 All sterile	None	Well
7	J. Z.	18	4 months	Well tolerated	70-120	In O.R., 120 colonies per c.c.	9/27/41 Ductal division	11 and 41 minutes post ligation—1 colony per c.c.	7 All sterile	None	Well
8	H. N.	9	5 weeks	Well tolerated	2-88	In O.R., heavily positive	1/9/42 Ductal ligation	11, 30, and 60 minutes post ligation— sterile	4 Sterile	None	Well
									8th day— 1 colony per 20 c.c.		
								2 and 7 hours —1 colony per 4 c.c.	6 Sterile		

*Case 1 is from the first series; Cases 5 to 8 are from the present series.

hemorrhage. The latter will be discussed fully later, but at this point it may be mentioned that this hazard has been reduced substantially by early operation and by the employment of a modification of operative technique which has been described in a separate communication.[12]

THE RATIONALE OF OPERATION

Abbott[1] has emphasized that the development of vegetations within the pulmonary artery is an early and practically constant feature of infected patent ductus arteriosus. This concept is of great importance from the standpoint of surgical therapy, for under the above circumstances it must be assumed that vegetations practically always are present within the pulmonary artery (and the ductus) at the time of operation. It is obvious that operation as performed—either ductal ligation or ductal division—does not result in removal of the aforementioned vegetations from contact with the circulating blood. Inasmuch as these surgical procedures have proved curative however, some explanation of their efficacy must be offered. Accordingly, a concept of the rationale of surgical treatment which has been set forth elsewhere[13] will be summarized at this point.

It is assumed that vegetations within the ductus and pulmonary artery are being constantly traumatized by the swirling ductal current, and that, as a result, infected emboli and organisms are being more or less uninterruptedly broken off into the circulation. Since the direction of the ductal current is continuously from the aorta toward the pulmonary artery, it is logical to maintain that infective material (derived from the vegetations mentioned above) for the most part enters the pulmonary artery.[15] However it is assumed that reverse eddy currents along the ductal wall also result in the entry of some material directly into the aorta. In the presence of an open ductus, there is a substantial increase in the pressure, volume, and rate of flow of blood in the pulmonary circuit. These in turn produce dilatation of the entire pulmonary arterial tree, and also create a powerful driving force which may facilitate the passage of infective material through the presumably already dilated capillaries of the lung.

It is postulated that, by closing the arteriovenous shunt, the traumatizing action of the swirling current upon the vegetations within the ductus and pulmonary artery, is terminated abruptly. This is assumed to result in a diminution in the amount of infective material which is broken off and enters the circulation. At the same time, none of the material now can enter the aorta directly through the ductus, but must enter the pulmonary artery exclusively. The factors responsible for dilatation of the pulmonary capillaries and for the increased driving power of blood in the pulmonary circuit also are eliminated by ductal ligation. This is assumed to result in an increase in the efficiency of the pulmonary protective (filtering and phagocytic) mecha-

nism. The relatively few organisms which still may pass through the capillaries of the lung, to enter the peripheral circulation, presumably are disposed of fairly readily; and the peripheral blood cultures become sterile quite promptly. Complete recovery, as evidenced by subsidence of clinical and laboratory evidences of infection, occurs only after the residual vegetations within the ductus and pulmonary artery heal. Whether such healing is due to alteration of local mechanical factors or nutrition of the vegetations is uncertain.

Evidence in support of the above-outlined concept of the mechanism of recovery will be found in the more detailed communication to which reference already has been made.[13] Regardless of whether or not our theory proves correct it remains an incontrovertible fact that, since all vegetations are not removed from the ductus and pulmonary artery at operation, *such vegetations heal spontaneously* in cases in which recovery ensues.

PATHOLOGIC FEATURES OF SURGICAL SIGNIFICANCE

The main pathologic features of *surgical* significance, in cases of infected patent ductus arteriosus complicated by subacute *Streptococcus viridans* endarteritis, were discussed in a previous communication.[17] These features, aside from the actual location of vegetations, are: (1) friability of the ductus, (2) adherence of the ductus to the surrounding structures (the most important of which is the pulmonary artery), and (3) dilatation and thinning of the pulmonary artery. Their importance is attributable to the fact that, as they become more marked, operation becomes more difficult and also more hazardous.

Friability is due to noninfectional (atheromatous and sclerotic) changes and superimposed infectional changes (endarteritis and arteritis) of the ductal wall. In children, noninfectional atheromatous and sclerotic changes are likely to be relatively slight, and the ductal wall usually is soft and nonresistant prior to the onset of infection. In adults however, even in the absence of infection, the ductal wall is more likely to be atheromatous and often is calcified in varying degree. When infection supervenes, the previously sclerotic wall becomes inflamed and often more friable. (At times, the infective process is so destructive that mycotic aneurysms of the ductus and pulmonary artery develop.) As will be shown presently, ductal friability appears in general to be proportional to (1) the age of the patient and (2) the duration and intensity of the local infective process.

Adherence of the ductus to the surrounding structures (the most important of which is the pulmonary artery) is due to periarteritis. The latter, in turn, usually is the result of an inflammatory process which begins in the intima and subsequently involves not only the wall of the ductus but the periductal tissues as well. Such periarteritis apparently is proportional to the intensity and duration of ductal infection.

Dilatation and thinning of the pulmonary artery appear to be proportional to the duration, diameter, and degree of patency, of the arteriovenous communication. Thus in adults (in whom the shunt has been of longer duration than in children), other factors being equal, dilatation of the pulmonary artery and thinning of the wall of the latter usually are proportionally much more marked than in younger persons.

THE RELATIONSHIP BETWEEN PATHOLOGIC CHANGES AND THE HAZARDS OF OPERATION

The degree of ductal friability and adherence and the extent of dilatation and thinning of the pulmonary artery have a direct bearing upon surgical therapy in so far as they tend to increase the difficulty of operation and the likelihood of accidental hemorrhage. Thus the separation of an extensively diseased, friable ductus from a greatly dilated, thin-walled pulmonary artery—when the two are firmly adherent—is a procedure which not only is difficult but dangerous. It is to be stressed that in cases of patent ductus arteriosus, infection supervenes most commonly *during adult life*. Under the latter circumstances even if the infection is of short duration (and the *infectional* ductal changes therefore slight), there still remains a great likelihood of encountering atheromatous and sclerotic (noninfectional) changes of the ductus and marked dilatation and thinning of the pulmonary artery, at operation. The dangers of operation in adults with patent ductus, even in the absence of superimposed infection, is well illustrated by a recent report of Gebauer and Nichol.[4] In their patient, a woman, aged 39 years, the diameter of the pulmonary artery was found at operation to be twice that of the aorta. The ductus was firmly adherent to the thin-walled dilated pulmonary artery, even though infection was not present. The ductus itself was short, and so sclerotic and calcific as to cast a recognizable ringlike shadow on the roentgen film. During the process of separating the ductus from the pulmonary artery, there was tremendous hemorrhage from the latter and from the aorta. Death occurred quickly.

In our first series, all four patients were adults between the ages of 24 and 63 years. The ductal and periductal changes, in general, were found to be more marked than in the cases of the present series. In three cases, hemorrhage occurred. In the remaining case, the ductal changes were milder and no hemorrhage took place.

In the four cases of the present series, the patients ranged in age from 9 to 51 years. The ductus was severely inflamed, markedly adherent, and friable in only one instance (Case 7). It is of importance to note that in this case, the infection not only was of the longest duration but clinically was the most severe. Slight bleeding occurred, but was controlled without great difficulty by clamping and then dividing the ductus completely. In two cases (Cases 5 and 6), the

TABLE II

CLINICOPATHOLOGIC FEATURES AS RELATED TO HEMORRHAGE AND THE RESULTS OF OPERATION*

CASE NO.	PATIENT	SEX	AGE	DURATION OF INFECTION	CLINICAL COURSE	ARTERITIS AND PERIARTERITIS	DILATATION OF P. A.	HEMORRHAGE	OPERATION	RESULT
1	S. S.	F	29	3 months	Mild, then septic	+++	++	Severe	Ductal division 1/27/40	Well
2	G. F.	F	29	5 weeks	Markedly septic	++	++	0	Ductal ligation 2/8/40	Unimproved. Died 8 months postoperative
3	A. S.	F	63	5 months	Mild. Slow deterioration	+++	+++	Fatal	3/16/40	Died
4	J. S.	F	24	3 months	Septic	+++	++	Fatal	4/8/40	Died
5	F. S.	F	29	Uncertain 31 (+) days. 6-8 weeks(?)	Mild. Slow deterioration	++	++	0	Ductal ligation 6/14/41	Well
6	G. O.	F	51	8 weeks	Mild. Slow deterioration	+	+++	0	Ductal ligation 9/4/41	Well
7	J. Z.	F	18	4 months	Markedly septic	+++	++	Slight	Ductal division 9/27/41	Well
8	H. N.	M	9	5 weeks	Mild	0	+	0	Ductal ligation 1/9/42	Well

*Cases 1 to 4 are from Series 1, and 5 to 8 from Series 2.

ductus was moderately firm and adherent. In these, the clinical course prior to operation was essentially one of gradual deterioration rather than of severe sepsis; and the infection was of appreciably shorter duration than in Case 7. In neither of these cases did bleeding occur. In the last case (Case 8), the ductus appeared quite normal, and operation was as simple as in cases of uninfected patent ductus. It is highly significant that (1) the patient was a child (the only nonadult in the group), (2) the infection was only of five weeks' duration, and (3) the clinical course was relatively mild.

As regards dilatation and thinning of the pulmonary artery, this feature was most marked in the oldest patients (in whom the arteriovenous shunt naturally had existed for the longest period). Thus in the original series, maximal enlargement was found in a patient aged 63, and in the second series, in a patient aged 51 (Case 6). In both, the pulmonary artery was so large as to obscure the aorta completely. The least dilatation was found in the youngest patient, a child of 9 (Case 8), in whom the diameter of the pulmonary artery was about equal to that of the aorta. In the remaining patients, the diameter of the pulmonary artery was somewhat greater than that of the aorta. Excluding the factor of the patient's age, the degree of dilatation and thinning of the pulmonary artery appears to be proportional to the diameter of the ductus and the completeness of ductal patency. In those instances in which the ductus is of large caliber and is widely patent, a substantial volume of aortic blood is shunted continuously into the pulmonary artery. As a result, the latter becomes considerably dilated. On the other hand, when the ductus is of small caliber or is only partially patent, enlargement of the pulmonary artery in general is likely to be less marked.

THE RELATIONSHIP BETWEEN DURATION OF INFECTION AND CURABILITY

When the infection is of relatively short duration, vegetations are likely to be confined to the ductus and pulmonary artery. Under such circumstances, ductal ligation would appear to have a fair chance of success. On the other hand, when infection is of longer duration, vegetations may spread to the cardiac valves or along the ductus directly into the aorta. In the presence of such vegetative foci, ligation of the ductus can not be expected to control the sepsis.

In view of great variation in the virulence of different strains of *Streptococcus viridans* and in the resistance of persons to such infection, there is considerable variation in the time which may elapse between the onset of infection and the spread of vegetations beyond the ductus and pulmonary artery. Therefore, it is impossible to state for how long a period an infection may be present and the case still remain operable. While in some cases, vegetations may not spread to the aorta or cardiac valves until relatively late, it must be remembered that in

the meantime such patients may succumb as the result of extensive extracardiac parenchymal disease (renal involvement, amyloidosis, etc.) induced by the prolonged sepsis.

In Cases 6, 7, and 8, the total duration of infection (from the time of onset of clinical manifestations to operation) was approximately eight weeks, four months, and five weeks, respectively. In Case 5 the duration of infection was impossible to determine with accuracy. The patient was admitted to the hospital because of a suspected fracture of the hip and, although fever and positive blood cultures were found, she had no complaint other than that of pain related to the trauma. She was observed in the hospital for thirty-one days prior to operation, and, accordingly, the duration of infection can be designated only as thirty-one (plus) days. However, her good physical condition and lack of symptoms on admission, together with the mildness of the inflammatory changes found at operation, suggested that the total duration of infection probably did not exceed six to eight weeks.

The rapidity with which blood cultures became negative and recovery ensued, in all four cases of this series, is the best indication of the effectiveness of operation in patients suffering from infection of relatively short duration. As a corollary, it may be stated that the longer the duration of infection, the less, in general, is the likelihood of cure.

THE DESIRABILITY OF EARLY OPERATION

From evidence already presented it is clear that operation, in patients suffering from early infection, is likely to be attended with little difficulty and danger and a promising outlook for recovery. Under the circumstances, it appears proper to warn against prolonged efforts to induce recovery by nonoperative methods. The most important of such forms of treatment is chemotherapy, with or without the use of certain adjuvants such as heparin or fever therapy. It is now generally agreed, by those with extensive experience in the use of sulfonamide drugs, that, if the latter are to prove effectual, they accomplish their purpose fairly promptly. Thus if intensive chemotherapy, in one form or another, does not result in the disappearance of clinical evidence of infection and sterilization of the blood stream within a period of two or three weeks, it is advisable to discontinue such treatment. Failure to observe this dictum leads to unnecessary delay which not only may render surgical therapy more difficult and hazardous, but also may militate against success even if operation is carried out successfully.

INDICATIONS AND CONTRAINDICATIONS TO OPERATION

Limitation of space prevents a complete discussion of the indications and contraindications to operation. However, at the present time, the author recognizes only two absolute operative contraindications. The

first is incontrovertible evidence that the open ductus is present as a compensatory mechanism for some other coexisting, congenital cardiovascular anomaly such as subaortic or pulmonic stenosis, coarctation of the aorta, etc. Ductal ligation, if carried out in the presence of one of the foregoing lesions, quickly leads to circulatory embarrassment and death. The second contraindication to operation is strong evidence that vegetations already have spread to the endocardium, aorta, or to the mitral or aortic valves. Under such circumstances, ductal ligation is of no value for the reason that the new lesions continue to feed infective material directly into the peripheral blood stream. [It is to be emphasized that the presence of an infection of long duration does not per se constitute a contraindication to operation, unless vegetations already have spread to the areas mentioned above. If extracardiac parenchymal changes alone have supervened, operation *probably* will prove of little value. However, no conclusive statement can be made in regard to the limitations of operation in such cases, and further experience may demonstrate that the latter are not all as hopeless as perhaps they appear. (The response of such cases to operation is being investigated by the author at the present time.)]

In a previous communication,[17] "poor condition of the patient" was stated to constitute a contraindication to operation. Further experience has demonstrated, however, that this statement requires some modification. "Poor condition" due to parenchymal disease has been discussed in the preceding paragraph and requires no further elaboration. However, poor condition due to other causes, such as cardiac failure or severe sepsis, merits additional consideration. The safety of operation in the presence of these complications is well illustrated by Case 7. This patient suffered not only from circulatory failure and severe sepsis, but also from a large pulmonary infarct. She was so acutely ill and also so debilitated that early fatality appeared imminent. On the assumption that there was little to be lost and perhaps much to be gained by prompt operation, the latter was performed after a three-day period of investigation and preparation. The response was so prompt and convalescence so uneventful as to be almost miraculous. In a somewhat less dramatic case, operation was well tolerated by a 51-year-old patient suffering from circulatory failure and pulmonary infarction (Case 6). In both of these cases, the difficulties of operation were further increased by the presence of extensive intrapleural adhesions.

OPERATIVE TECHNIQUE

Cyclopropane or ethylene anesthesia, administered through a snugly fitting face mask, was employed in all cases. (The intratracheal method not only is unnecessary but often induces severe laryngotracheal irritation which in turn may lead to serious pulmonary complications.)

In the three cases in which ligation was performed, the ductus was tied off with two heavy silk ligatures. In the case in which complete division was performed (Case 7), two Carmalt clamps were applied and the ductus severed between them. Each stump then was ligated with a single heavy silk ligature, no attempt being made to suture the ductal stumps or to invert them. The mediastinal pleura was left open in all cases except the last (Case 8). There, because of the presence of minimal inflammatory changes in the mediastinal tissues, the pleura was closed loosely.

The modified technique of freeing the ductus has already been described[12] and will not be detailed at this point. Suffice it to state that it first consists of freeing an area of the aortic arch, immediately adjacent to the proximal end of the ductus. This dissection is carried out *within the adventitia* of the aorta, and *not outside* the latter. After a "tunnel" has been made on the posteroinferior surface of the aortic arch, within the substance of the adventitia, the interadventitial plane of cleavage is carried downward and mesially behind the ductus. In this manner, by freeing a layer of ductal adventitia (which is retracted away from the ductus together with the pulmonary artery), the ductus is separated from the latter structure. (In the presence of periarteritis, the layers of the ductal adventitia can be separated from one another more readily than the ductus, as a whole, can be separated from the pulmonary artery.) With good illumination and the use of small retractors, the relatively "blind" dissection of the posteromesial ductal wall can be carried out under fairly complete visualization.

With the employment of this technique, the time of operation has been reduced substantially and the procedure appears to have been rendered more safe. Slight bleeding occurred in Case 7 after the ductus had been practically completely freed. However, in this case, the ductus was so adherent to the pulmonary artery that it is doubtful whether the two structures could have been separated safely by the usual method. In Cases 5 and 6 the ductus was adherent, but to a lesser degree than in Case 7. Although the dissection might have been carried out successfully by the usual method, the ease and safety with which it was performed in these two cases was striking. (In Case 6, operation was performed in fifty-five minutes.) Case 8, because of minimal local inflammatory changes, could not be considered a fair test.

THE RESULTS OF OPERATION

All four patients recovered following operation, and all have remained well. (Up to the present time,* Case 5 has been followed for one year, Case 6, for nine and one-half months, Case 7, for eight and one-half months, and Case 8, for five months.) These results are to be contrasted with those obtained in our original series (one cure in four

*June 15, 1942.

cases). It is to be emphasized again that three patients received no chemotherapy following operation. One received an insignificant amount of sulfapyridine after all postoperative blood cultures, taken up to that time, had been reported negative.

While this report is concerned primarily with personal experiences in the surgical treatment of infected patent ductus arteriosus, it seems proper to set forth briefly the results which have been achieved by others. Since presenting our first successful case in June, 1940, only two other reports of operative cases of infected patent ductus have appeared in the literature.[2, 11] The first was by Keele and Tubbs,[11] of London, in July, 1940; and at the time of publication (six months postoperatively) the patient's blood cultures still were *positive.* A more recent report concerning this patient discloses that, following intensive chemotherapy, the blood cultures finally became sterile *thirteen months* after operation.[2] In view of the long interval between operation and recovery, it might be maintained quite properly that the latter was due solely to the use of chemotherapy. The second case was reported by Bourne, Keele and Tubbs,[2] fairly recently. In this case, the use of sulfapyridine resulted in sterilization of the blood stream *prior to operation.* Furthermore, the administration of the drug was continued after operation. Under these circumstances, it might be claimed with some justification that the use of chemotherapy actually had resulted in cure prior to operation, or at least that it was partially responsible for recovery after operation. Accordingly, it is impossible to evaluate the individual role of surgery or chemotherapy in the recovery of this patient.

Gross[8] states that he has operated upon four cases of infected patent ductus arteriosus. In the first case, following ligation and chemotherapy, infection continued. (The patient had been ill for eleven months prior to operation.) In the next two cases, chemotherapy rendered the blood cultures sterile *prior to operation.* Following ductal ligation, chemotherapy was continued. Both of these patients recovered. The same objections that might be raised in regard to the case of Bourne, Keele, and Tubbs, also would apply in these two cases.

In Gross' last case, a blood culture was *positive* on the day prior to operation. Ductal ligation, followed by the administration of chemotherapy, resulted in recovery. Humphreys,[10] of New York, has operated upon two cases of infected patent ductus arteriosus. As in Gross' last case, the blood cultures were positive prior to operation in spite of intensive chemotherapy. Ductal ligation, followed by further chemotherapy, resulted in recovery in both instances. In the aforementioned three cases, in view of the failure of chemotherapy to sterilize the blood stream *preoperatively,* recovery *cannot* be ascribed solely to the use of chemotherapy *following operation.* On the other hand, recovery cannot be attributed entirely to surgery, for the administration of chemo-

therapy after operation served to introduce an additional therapeutic factor. However, it may be concluded that the combination of operative treatment and chemotherapy was responsible for recovery, and that these measures constitute an effective form of treatment of subacute bacterial endarteritis superimposed on patent ductus arteriosus. The author, by avoiding the use of chemotherapy following operation, demonstrated originally that ductal division by itself was capable of bringing about recovery. As the result of additional personal experience, he is able to confirm that observation and to add that ductal ligation is similarly effective.

SUMMARY

Various features of infected patent ductus arteriosus, which bear a relationship to surgical therapy, are discussed.

Four additional patients suffering from subacute *Streptococcus viridans* endarteritis superimposed on patent ductus arteriosus have been subjected to operation.

Chemotherapy was administered in each case prior to operation without resulting in sterilization of the blood stream.

Operation consisted of ductal ligation in three instances and of ductal division in one.

Following operation, three of the patients received no chemotherapy. The remaining patient received a total dose of 10 Gm. of sulfapyridine (on the twelfth postoperative day) because of continuing fever of obscure etiology. However, all postoperative blood cultures—six in number—already had been reported negative before chemotherapy was administered.

All four patients recovered and have remained free of infection since operation.

Among eight cases, comprising the first and second series, there were five cures, two deaths from accidental hemorrhage, and one failure ascribable to the presence of vegetations upon the aortic valve at the time of operation. Thus among six operative *survivals,* there were five recoveries and one failure (attributable to improper selection of the case for operation).

CONCLUSIONS

Surgical therapy, consisting of ductal ligation or division, is an effective form of treatment of subacute *Streptococcus viridans* endarteritis superimposed on patent ductus arteriosus.

It is not necessary to supplement such treatment with chemotherapy.

Operation is most likely to be attended with low morbidity and mortality and a high incidence of cure, if operation is performed during the early stages of infection.

ADDENDUM

Since preparing this paper for publication, two additional patients suffering from patent ductus arteriosus complicated by infection, have been cured by operation.

REFERENCES

1. Abbott, M. E.: Atlas of Congenital Cardiac Disease, New York, 1936, American Heart Association.
2. Bourne, G., Keele, K. D., and Tubbs, O. S.: Ligation and Chemotherapy for Infection of Patent Ductus Arteriosus, Lancet **241**: 444, 1941.
3. Chester, W.: Patent Ductus Botalli With Subacute Bacterial Endocarditis and Recovery, Am. Heart J. **13**: 492, 1937.
4. Gebauer, P. W., and Nichol, A. D.: Ligation of the Patent Ductus Arteriosus, Ohio State M. J. **37**: 538, 1941.
5. Graybiel, A., Strieder, J. W., and Boyer, N. H.: An Attempt to Obliterate the Patent Ductus Arteriosus in a Patient With Subacute Bacterial Endarteritis, Am. Heart J. **15**: 621, 1938.
6. Gross, R. E.: A Surgical Approach for Ligation of a Patent Ductus Arteriosus, New England J. Med. **222**: 510, 1939.
7. Gross, R. E.: Surgical Management of the Patent Ductus Arteriosus, Ann. Surg. **110**: 321, 1939.
8. Gross, R. E.: Personal Communication.
9. Gross, R. E., and Hubbard, J. P.: Surgical Ligation of a Patent Ductus Arteriosus: Report of First Successful Case, J. A. M. A. **112**: 729, 1939.
10. Humphreys, G. H.: Personal Communication.
11. Keele, K. D., and Tubbs, O. S.: Combined Ligation of Ductus Arteriosus and Sulfapyridine Therapy in a Case of Influenzal Endarteritis, St. Bart. Hosp. War Bull. **1**: 175, 1940.
12. Touroff, A. S. W.: A Modified Technique of Surgical Ligation of Patent Ductus Arteriosus, Surgery **12**: 24, 1942.
13. Touroff, A. S. W.: The Rationale of Operative Treatment of Subacute Bacterial Endarteritis Superimposed on Patent Ductus Arteriosus, Am. Heart J. **23**: 847, 1942.
14. Touroff, A. S. W.: The Results of Surgical Treatment of Patent Ductus Arteriosus Complicated by Subacute Bacterial Endarteritis, Am. Heart J. (To be published.)
15. Touroff, A. S. W.: Blood Cultures From the Pulmonary Artery and Aorta in a Patient With Infected Patent Ductus Arteriosus, Proc. Soc. Exper. Biol. & Med. **49**: 568, 1942.
16. Touroff, A. S. W., and Tuchman, L. R.: Subacute Streptococcus Viridans Endarteritis Superimposed on Patent Ductus Arteriosus. Spontaneous Recovery. Recurrence After Twelve Years. Recovery Following Surgical Treatment, Am. Heart J. **23**: 857, 1942.
17. Touroff, A. S. W., and Vesell, H.: Experiences in the Surgical Treatment of Subacute Streptococcus Viridans Endarteritis Complicating Patent Ductus Arteriosus, J. THORACIC SURG. **10**: 59, 1940.
18. Touroff, A. S. W., and Vesell, H.: Subacute Streptococcus Viridans Endarteritis Complicating Patent Ductus Arteriosus: Recovery Following Surgical Treatment, J. A. M. A. **115**: 1270, 1940.
19. Touroff, A. S. W., Vesell, H., and Chasnoff, J.: Operative Cure of Subacute Streptococcus Viridans Endarteritis Superimposed on Patent Ductus Arteriosus. Report of the Second Successful Case, J. A. M. A. **118**: 890, 1942.

THE SURGICAL ANATOMY OF THE PULMONARY LOBES

EDWARD M. KENT, M.D. (BY INVITATION), AND BRIAN BLADES, M.D.
ST. LOUIS, MO.

THE careful dissection and individual disposal of hilar structures have replaced all other methods for total pneumonectomy except in those few instances in which the pursuit of such a technique has been found impossible. Although of more recent application and less firmly grounded upon clinical experience, the same surgical principles of individual management of anatomic structures promise to become equally dominant as the preferable method of performing lobectomy[1, 3] or segmental resection.[2]

The anatomic patterns of the hilum of the lungs have been clearly delineated[4, 6, 7] so that surgical application of this knowledge has become widespread. The surgical anatomy of the roots of the lower lobes[1, 3] and of the right middle lobe and the lingula of the left upper lobe[2, 3] have also been described.

There has been, however, no description of the surgical anatomy of the roots of the upper lobes and there has appeared no composite, coordinated picture of the entire problem as it applies to pulmonary resection. It was in an effort to fulfill this need that the present investigation was pursued and the observations which have been made are based upon anatomic studies of 227 lungs dissected immediately post mortem, the study of the intrathoracic structures in seven embalmed cadavers and the clinical application of this knowledge in twenty-seven instances of lobectomy.

It is probable that the use of a familiar structural framework to which we may add the additional important factors will greatly facilitate the description. The tracheobronchial tree is well known to those who are concerned with intrathoracic disease and, therefore, will be discussed first and the anatomic relationships of the blood vessels will be oriented accordingly.

In performing total pneumonectomy the surgeon is concerned with the right and left main bronchi (Fig. 1) and in lobectomy he is interested in the principal divisions of the main bronchi (Fig. 1). On the right, these consist of the upper lobe bronchus, the middle lobe bronchus, and the lower lobe bronchus with its important branch to the superior

From the Chest Service of the Barnes Hospital and the Department of Surgery, Washington University School of Medicine.

Read (by title) before the Twenty-Fourth Annual Meeting of the American Association for Thoracic Surgery at Toronto, Canada, June 9, 10, and 11, 1941.

division of the lobe. On the left, we must concern ourselves with the upper lobe bronchus and its subsidiary to the lingula, with the lower lobe bronchus, and, as on the right, with the branch bronchus to the superior division of the lower lobe.

The right upper lobe bronchus has been found to be of sufficient length to allow division as a unit and careful closure in the great majority of instances. The right middle lobe bronchus is always of sufficient length to allow easy individual management. Churchill[3] has expressed his belief that intrahilar resection of the middle lobe can be performed, but that it is unwarranted because the tourniquet leaves a hilar stump so small that it rarely results in complications. The fissure between the right upper and middle lobes is incomplete or absent in a high percentage of instances[1] and is the principal obstacle to excision of either of these structures by individual ligation.

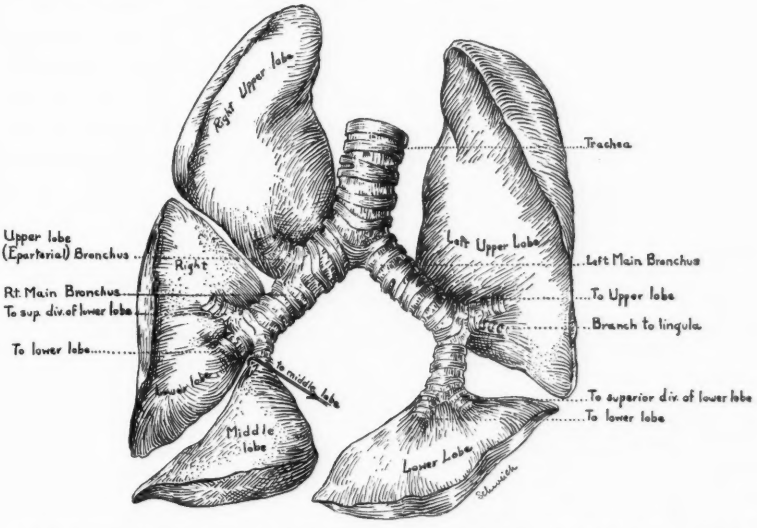

Fig. 1.—Illustration made from fresh specimen. The fissures have been completed and the lobes retracted from normal position to make it possible to visualize all points under discussion; the levels of origin of the right middle lobe bronchus and the branch bronchus to the superior division of the right lower lobe are to be emphasized. Proportions are incorrect, especially the pulmonary lobes which have partially collapsed.

The right lower lobe bronchus is, for surgical purposes, short at best because its superior division branch arises at a level in close proximity to that of the middle lobe bronchus; indeed, it is often at the same level and rarely even higher. For these reasons it is frequently necessary to divide the main trunk of the right lower lobe bronchus and its superior division branch separately, and to effect a separate closure of each. It has been suggested[3] that the two orifices may be made one before repair

is attempted but we can see no possible advantage in making one larger bronchial opening of two smaller structures only to be forced to attempt to accomplish with suture material that which we have destroyed with the scalpel. The bronchus to the superior division of the lower lobe is also important because of its role in segmental resection, whether it is to be preserved or removed in such a plan of attack upon localized disease of the lower lobes.

The bronchial divisions on the left side which must be considered are the upper lobe bronchus, the bronchial division to the lingula of the upper lobe, and the lower lobe bronchus with its branch to the superior division of the lobe.

The left upper lobe bronchus is of adequate length to allow direct surgical attack upon it and to make subsequent closure practicable. The bronchial division to the lingula of the left upper lobe is important because of the frequency with which this segment of the lobe must be resected in the surgical treatment of bronchiectasis. Segmental resection of the lingula as advocated by Churchill and Belsey[2] is predicated upon the individual management of its bronchus and of its venous and arterial components. The anatomic pattern of the bronchus to the lingula is of two chief types (Fig. 2), and is suitable for direct surgical attack in either instance.

Fig. 2.—Diagrammatic representation of variations in the mode of origin of the branch bronchus to the lingula and the axillary division of the upper lobe bronchus; the pattern shown in *A* is the more common. (Churchill and Belsey, Ann. Surg. **109**: 481, 1939.)

The left lower lobe bronchus differs from its counterpart on the right side in that there is no middle lobe bronchus to compromise its length and hence surgical management is distinctly less difficult. On the left side the branch bronchus to the superior division of the lower lobe is of significance because of its role in segmental resection of a part of the lobe.

In summary, it is possible to declare that the bronchi of all lobes are of adequate proportions to allow direct surgical attack. In addition, the bronchial divisions to the lingula of the left upper lobe and the branch bronchi to the superior divisions of the lower lobes are adequate for similar management in segmental resection. As has been pointed out, in

the performance of right lower lobe resection by the individual ligation technique, it is often necessary to divide separately the main trunk of the lower lobe bronchus and its superior division branch. This anatomic characteristic constitutes the chief technical hazard of the method but it may be overcome by careful dissection. Furthermore, observation of the levels of these components may be made at preoperative bronchoscopy and thus the surgeon may be forewarned concerning the anatomic pattern likely to be encountered.

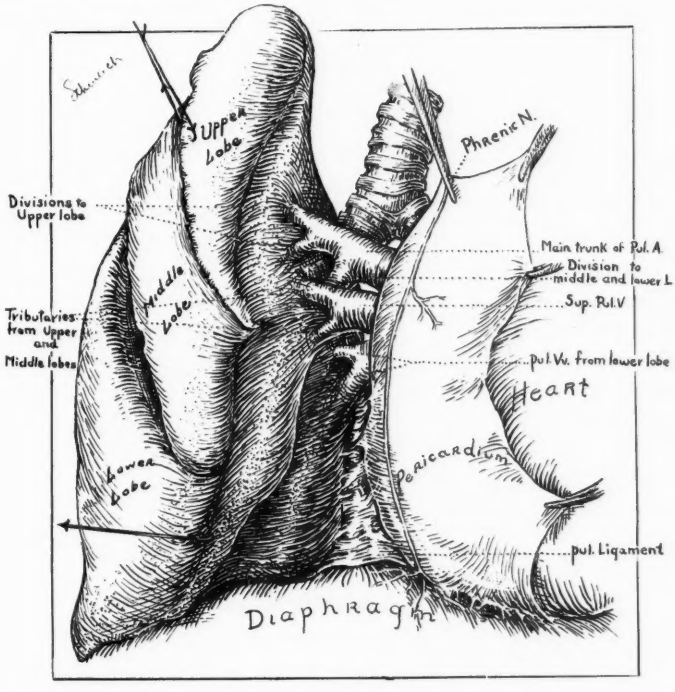

Fig. 3.—Illustration made from dissection of fresh specimen, anterior aspect right side; the relative positions of the pulmonary veins, the pulmonary artery, and the right main bronchus are shown and the position of the inferior pulmonary vein and its relationship to the upper end of the inferior pulmonary ligament are emphasized.

The pulmonary veins and their major lobar tributaries will be considered next. These structures are two in number on each side and all empty into the left auricle. They are found immediately beneath the pleura which covers the hilum and may be approached with facility by an anterior initiation of the dissection.

The venous pattern for the right side may be seen in Fig. 3. The superior pulmonary vein lies in an anterior position in the hilum and receives tributaries from the upper and middle lobes, a fact which must be borne in mind when resection of either of these lobes is contemplated.

If the entire vein is sacrificed during the resection of one of these lobes, the other lobe has been deprived of its venous channel and may not survive. Fig. 4 is an illustration of the anterior view of the hilum in which the distribution of the venous tributaries is clearly depicted. Rarely, the venous tributary from the middle lobe enters directly into the left auricle and thereby exists as a third right pulmonary vein.

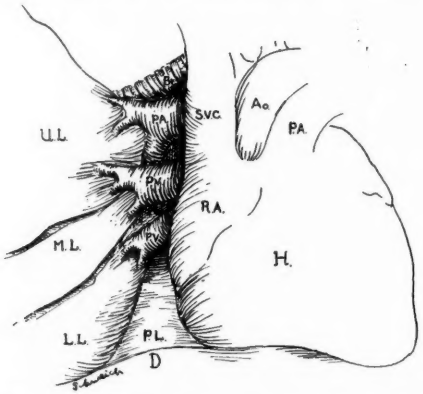

Fig. 4.—Diagrammatic illustration of anterior view of right pulmonary hilum. *U.L.*, upper lobe; *M.L.*, middle lobe; *L.L.*, lower lobe; *Br.*, right main bronchus; *P.V.*, pulmonary veins; *P.L.*, inferior pulmonary ligament; *D.*, diaphragm; *S.V.C.*, superior vena cava; *Ao.*, aorta; *P.A.*, pulmonary artery; *R.A.*, right auricle, and *H.*, heart.

The right inferior pulmonary vein collects the aerated blood of the right lower lobe and enters the left auricle just below the point of entry of the superior pulmonary vein. This vein is situated immediately anterior to the hilar end of the inferior pulmonary ligament and may be found with accuracy by carefully dividing the ligament, or by preserving the ligament as a guide and initiating the dissection of the vein between the posterior wall of the vessel and the anterior surface of the ligament. The first tributary of the inferior pulmonary vein comes from the superior division of the lower lobe and this may be preserved or divided, depending upon which portions of the lobe are to be excised by segmental resection.

The left pulmonary veins are also two in number and are best approached from the anterior aspect of the hilum (Fig. 5). The left superior pulmonary vein arises in the left upper lobe and, at the lobe root, is formed by the union of three or four principal tributaries. One of these tributaries arises in the lingula and is important in segmental resection of this part of the upper lobe. As on the right side, the left inferior pulmonary vein arises in the lower lobe and empties into the left auricle. The similarity of this vessel to its counterpart on the right is clearly indicated in Fig. 6. This vein is also just anterior to the hilar end of the inferior pulmonary ligament and may be dealt with as

described for the right inferior pulmonary vein. The first tributary of this vessel arises in the superior division of the lobe and the implications of this fact have been alluded to in the discussion of the right side.

The pulmonary arteries are to be considered at this point and it may be recalled that the right pulmonary artery is found anterior to and at a slightly lower level than the right main bronchus and is posterior to and slightly above the superior pulmonary vein (Figs. 3 and 4). An illustration of the course and of the main branches of the right

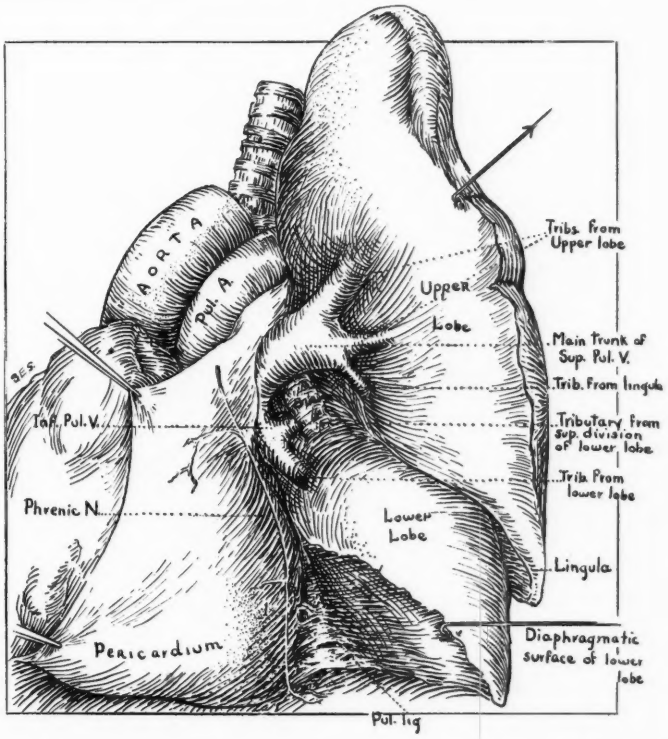

Fig. 5.—Illustration made from dissection of fresh specimen, anterior aspect pulmonary hilum on left side; the relative positions of the pulmonary veins, the pulmonary artery, and the left main bronchus are shown.

pulmonary artery is provided in Fig. 7. In this sketch the middle lobe is shown to be deflected anteromedially so that the entire course of the artery and all its branches may be depicted. The first branch of the right pulmonary artery is that to the upper lobe (Figs. 3, 4, and 7) and this in turn divides into two or more channels of considerable size before disappearing into the lobe. The next branch of the right pulmonary artery is the division to the middle lobe (Fig. 7), and this may be double (Fig. 8) rather than the more common finding of a single

vessel. The first branch arising below the level of the middle lobe artery is that to the superior division of the lower lobe (Figs. 7 and 8); thereafter, the artery continues into the lobe to supply the remaining segments. Just as in the case of the bronchus, the arterial division to the lower lobe may arise at or near the level of origin of the middle lobe artery (Fig. 8), a fact which must be borne in mind. It is worth while to note that the arterial pattern at this point usually indicates the bronchial pattern to be encountered.

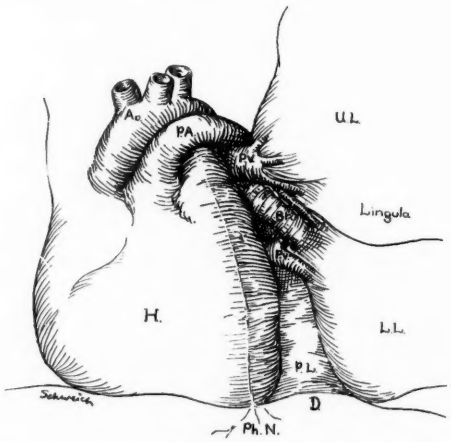

Fig. 6.—Diagrammatic illustration left pulmonary hilum, anterior aspect; relationships of major structures defined. *U.L.*, upper lobe; *L.L.*, lower lobe; *Ao.*, aorta; *P.A.*, pulmonary artery; *P.V.*, pulmonary veins; *Br.*, lower lobe bronchus; *P.L.*, inferior pulmonary ligament; *D.*, diaphragm; *Ph.N.*, left phrenic nerve; and *H.*, heart.

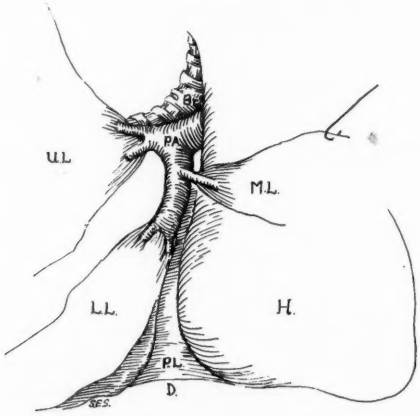

Fig. 7.—Diagrammatic illustration of the distribution of the right pulmonary artery; middle lobe has been retracted anteromedially and there is a single arterial division to the middle lobe in this instance. *Br.*, right main bronchus; *U.L.*, upper lobe; *M.L.*, middle lobe; *L.L.*, lower lobe; *P.A.*, right pulmonary artery; *P.L.*, inferior pulmonary ligament; *D.*, diaphragm; *H.*, heart. The veins have not been included so that clarity may be increased.

The most common anomaly encountered in the study of the right pulmonary artery has been the presence of a branch to the upper lobe which arises at or below the level of the artery to the superior division of the lower lobe, but this branch is usually small and its ligation is of no consequence.

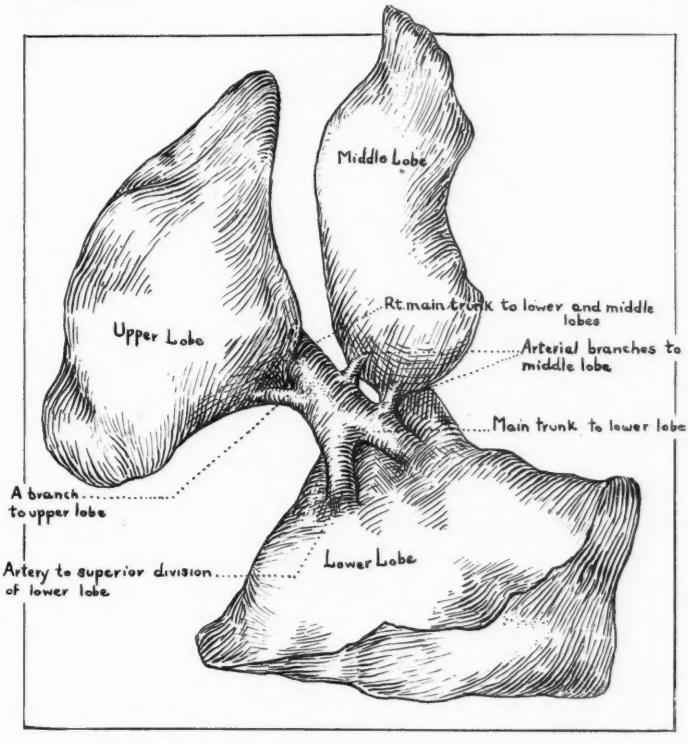
Middle Lobe
Upper Lobe
Rt. main trunk to lower and middle lobes
Arterial branches to middle lobe
Main trunk to lower lobe
A branch to upper lobe
Artery to superior division of lower lobe
Lower Lobe

Fig. 8.—Diagrammatic illustration of distribution of right pulmonary artery excluding branch to upper lobe; the chief interest lies in the double supply to the middle lobe, which was found in about 25 per cent of dissections.

While the portion of the right pulmonary artery which is of importance in pulmonary resection remains anterior to or anterolateral to the bronchi, the left pulmonary artery does not do so. This radical difference between the two arteries is of great surgical importance, as will be shown.

The left pulmonary artery at first lies anterior to the left main bronchus, but soon ascends over the superior surface of the main bronchus to pass around and behind the left upper lobe bronchus (Fig. 9), thereby entering the interlobar fissure, where it remains. As the artery passes around and behind the upper lobe bronchus it gives off several short ramifications (Figs. 9 and 10) which supply the major portion of the upper lobe. These short branches begin as soon as the

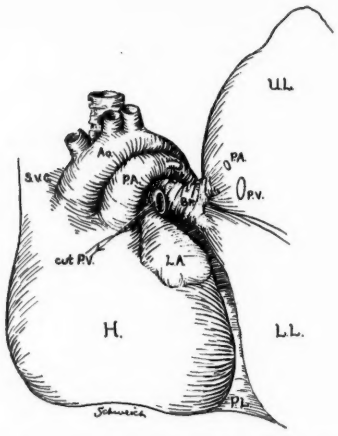

Fig. 9.—Diagrammatic illustration of distribution of left pulmonary artery as seen from the anterior aspect. Note how artery ascends over main bronchus to disappear around and behind upper lobe bronchus; the first arterial branch to the upper lobe has been divided and the veins have been eliminated for clarity. *U.L.*, upper lobe; *L.L.*, lower lobe; *Ao.*, aorta; *P.A.*, pulmonary artery; *S.V.C.*, superior vena cava; *P.V.*, auricular end of severed superior pulmonary vein; *Br.*, left main bronchus; *L.A.*, left auricle; *P.L.*, inferior pulmonary ligament; and *H.*, heart.

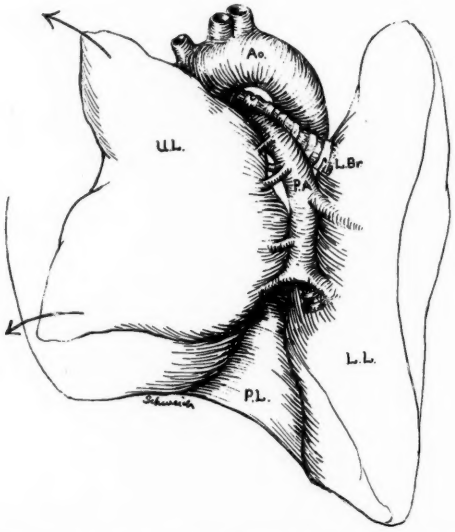

Fig. 10.—Diagrammatic illustration of pattern of left pulmonary artery as seen in the interlobar fissure. Note the several short branches to the upper lobe as the pulmonary artery passes around and behind upper lobe bronchus; the arterial branch to the superior division of the left lower lobe arises at a level above the point of origin of the artery to the lingula in this specimen. *Ao.*, aorta; *U.L.*, upper lobe; *L.L.*, lower lobe; *L.Br.*, branch bronchus to superior division left lower lobe; *P.A.*, left pulmonary artery; and *P.L.*, inferior pulmonary ligament.

artery approaches the upper lobe and they vary from four to seven in number. In resecting the left upper lobe by individual ligation or intrahilar dissection, each of these short vessels must be dissected, ligated, and divided separately and the main trunk of the artery must not be obstructed in order that the lower lobe will survive. Although we have not had the opportunity to attempt resection of the left upper lobe by this method, we are convinced that it will prove to be hazardous, if not technically impossible, in most instances.

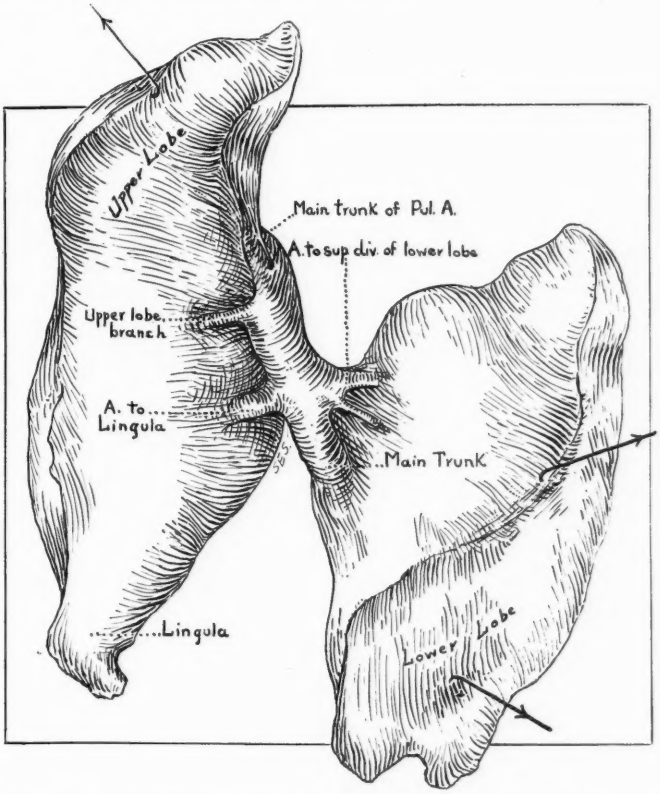

Fig. 11.—Diagrammatic illustration of distribution of left pulmonary artery as it lies deep in the left interlobar fissure; in this specimen the arterial supply to the superior division of the lower lobe is double and the point of origin is opposite that of the arterial branch to the lingula.

The remainder of the left pulmonary artery courses inferiorly and anteriorly deep into the interlobar fissure and gives rise to the branch to the superior division of the lower lobe and the branch to the lingula of the left upper lobe before entering the lower lobe to divide into its numerous terminal branches (Figs. 10 and 11). As can be seen in Figs. 10 and 11, the branch to the superior division of the left lower lobe may

arise opposite (Fig. 11) or above (Fig. 10) the level of origin of the branch to the lingula. These latter observations are of great importance in resection of the lower lobe, for each of these two main branches to the lobe must be disposed of separately so that the branch to the lingula may be preserved. The branch of the left pulmonary artery which supplies the lingula (Figs. 10 and 11) is important in resection of that portion of the upper lobe.

SUMMARY OF RELATIONSHIPS OF THE STRUCTURES OF THE PULMONARY LOBES

In the case of the right upper lobe the vein is anterior, the artery immediately posterior and slightly above the vein, and the bronchus is posterior and somewhat superior to the artery.

The vein to the middle lobe is anterior, the bronchus posterior to the vein, and the artery posterior to the bronchus.

While the inferior pulmonary vein is best approached from the anterior aspect, it actually enters the lobe posterior to the bronchus. The artery is found in the interlobar fissure just lateral and anterior to the bronchus.

In the case of the left upper lobe, the vein is anterior as on the right side, but the artery, while at first lying anterior to the main bronchus, soon passes behind and around the upper lobe bronchus to reach the interlobar fissure and numerous short branches spring from the main trunk as it pursues this route.

The relationships of the components of the left lower lobe root are similar to those on the right side. The vein, although best attacked from the anterior aspect, actually enters the lobe just posterior to the bronchus and the artery is found in the interlobar fissure close to and intimately associated with the anterolateral aspect of the bronchus.

The bronchial arteries require comment, although they are not of great surgical importance. They arise in several different manners, including: by a common trunk from the aorta, separate aortic origins, and origin from the first intercostal artery. While they are said to be found overlying the membranous posterior wall of the main bronchus, our observations indicate that there is great variation in their relationship to the main bronchus and to the branch bronchi. They are small vessels and an attempt to identify and ligate them is unwarranted since they may be easily dealt with in the usual fashion when cut.

We believe that a truly complete fissure does not exist in the human being,[1] not, at least, in the sense that it does in the dog. There is always some degree of confluence of tissues of adjacent lobes in the immediate vicinity of the bronchial axis and, for statistical purposes, we consider those fissures showing no more than a slight degree of such union to be complete. The fissure between the right middle and upper lobes has been

found to be absent in 21 per cent of the specimens and incomplete in 67 per cent. We found no instances of absence of fissures between the upper and lower lobes, although incompleteness was observed in about 30 per cent of the specimens on each side. This incompleteness is most marked between the superior division of the lower lobe and the adjacent inferior surface of the upper lobe. It is quite rare to find an incomplete fissure between the right middle and lower lobes (12.5 per cent).

Marked variations from what we conceive to be the normal anatomy have not been seen in our work. We have mentioned some of the minor differences in vascular pattern which we have encountered. MacCready[5] has reported two instances in which he found the left superior pulmonary vein entering the left innominate vein and one cadaver in which the right superior pulmonary vein entered directly into the superior vena cava.

Another source of minor anatomic anomalies has to do with the presence of marked or complete fissures where one does not expect to find them. This applies chiefly to an abnormally discrete delineation of the lingula or of the superior divisions of the lower lobe. We have seen only one instance when this state produced a striking abnormality and in that specimen a very large superior division of the left lower lobe was completely delineated by fissures, was supplied by a large bronchus, and generally appeared as a third left pulmonary lobe. Nevertheless, we have been impressed with the high degree of uniformity of anatomic pattern found in these studies.

COMMENT

Anatomic studies and clinical experiences[1, 3] have indicated that pulmonary resection by the individual ligation technique may be applied successfully in total or partial removal of the lower lobes, in excision of the lingula of the left upper lobe, and in right middle lobe lobectomy. The anatomy of the right upper lobe indicates that this lobe may be similarly dealt with when resection is to be performed and in at least one instance[8] this has been performed.

Studies of the anatomy of the left upper lobe have led us to conclude that, except under unusually favorable circumstances, the individual ligation technique for resection will be hazardous or impossible. It is to be borne in mind that the relationships of the left pulmonary artery are such that it may be occluded during left upper lobe resection by any method.

Resection of the right lower and middle lobes is often required in the surgical treatment of bronchiectasis. We believe that, if the fissures can be established, these two lobes may be removed together by the individual ligation technique. This can be accomplished by dealing with the following structures: the right inferior pulmonary vein, the branch of the superior pulmonary vein arising in the middle lobe, the right

pulmonary artery just above its branch to the middle lobe, and the right bronchus at a point slightly above the level of the middle lobe bronchus.

REFERENCES

1. Blades, B., and Kent, E. M.: Individual Ligation Technique for Lower Lobe Lobectomy, J. THORACIC SURG. **10**: 84, 1940.
2. Churchill, E. D., and Belsey, R.: Segmental Pneumonectomy in Bronchiectasis, Ann. Surg. **109**: 481, 1939.
3. Churchill, E. D.: Resection of the Lung, Surgery **8**: 961, 1940.
4. Crafoord, Clarence: On the Technique of Pneumonectomy in Man, Acta chir. Scandinav. (Suppl. 54) **81**: 1, 1938.
5. MacCready, P. B.: Anomalies of the Pulmonary Veins, Bull. Johns Hopkins Hosp. **29**: 271. 1918.
6. Mason, G. A.: Extirpation of Lung (Hunterian Lecture Abridged), Lancet **1**: 1047, 1936.
7. Reinhoff, W. F., Jr.: Pneumonectomy: A Preliminary Report of the Operative Technique in Two Successful Cases, Bull. Johns Hopkins Hosp. **53**: 390, 1933.
8. Butler, E. F.: In discussion of reference No. 1, Blades and Kent.

CARDIAC ARRHYTHMIAS AND OTHER ELECTROCARDIOGRAPHIC CHANGES DURING EXPERIMENTAL PNEUMONECTOMY

HENRY L. CABITT, M.D., MARK D. ALTSCHULE, M.D. (BY INVITATION), AND NORMAN ZAMCHECK, A.B. (BY INVITATION)
BOSTON, MASS.

SUDDEN death without apparent cause occasionally occurs in man during the course of operations on the lungs. Since such an event suggests the onset of some form of abnormal cardiac function, it was considered desirable to study the effect of pneumonectomy on cardiac action.

MATERIAL AND METHODS

Eleven dogs ranging in weight from 14 to 55 pounds were used. Anesthesia was induced by the intravenous injection of a 6 per cent solution of pentobarbital sodium in doses of 0.2 c.c. per pound of body weight. Artificial respiration through a tracheal cannula was maintained during the operation. Electrocardiograms were taken with needle electrodes before and at frequent intervals during pneumonectomy. The second lead was employed throughout.

RESULTS

In eight of eleven experiments, manipulation of the main branch of the pulmonary artery at the hilus or its lobar subdivisions immediately caused changes in the T waves. In four of the experiments manipulation of a pulmonary vein also caused changes in the T wave. In most cases the T wave became inverted (Fig. 1, *a* and *b*), but when the T wave was inverted initially, it became upright following manipulation (Fig. 1, *c* and *d*). In three instances ligation of a lobar, or of a larger pulmonary vein, resulted in the appearance of a split P wave (Fig. 1, *e*).

During the course of five operations, cardiac arrhythmias occurred as the hilus of the lung was divided. In three experiments frequent auricular premature beats occurred; in one of these experiments, a shift of the pacemaker was apparent (Fig. 2, *A*). In two other experiments more serious arrhythmias were noted, namely, numerous ventricular extrasystoles arising from different foci and almost entirely replacing the normal beats over a period of thirty seconds (Fig. 2, *B*) and very marked sinus bradycardia with occasional auricular premature beats (Fig. 2, *C*); in both instances the changes were transitory.

From the Medical Research Laboratories and the Thoracic Clinic, Beth Israel Hospital, the Department of Medicine, Harvard Medical School, and the Department of Surgery, Tufts Medical School.

This study was aided by a grant from the Charlton Fund.

Read (by title) before the Twenty-Fourth Annual Meeting of the American Association for Thoracic Surgery at Toronto, Canada, June 9, 10, and 11, 1941.

DISCUSSION

Electrocardiographic changes occurred during pneumonectomy in all dogs used in this study. In most instances the changes were limited to the T waves and were similar to those described by other authors as occurring following trauma to pulmonary blood vessels.[1-6] Although

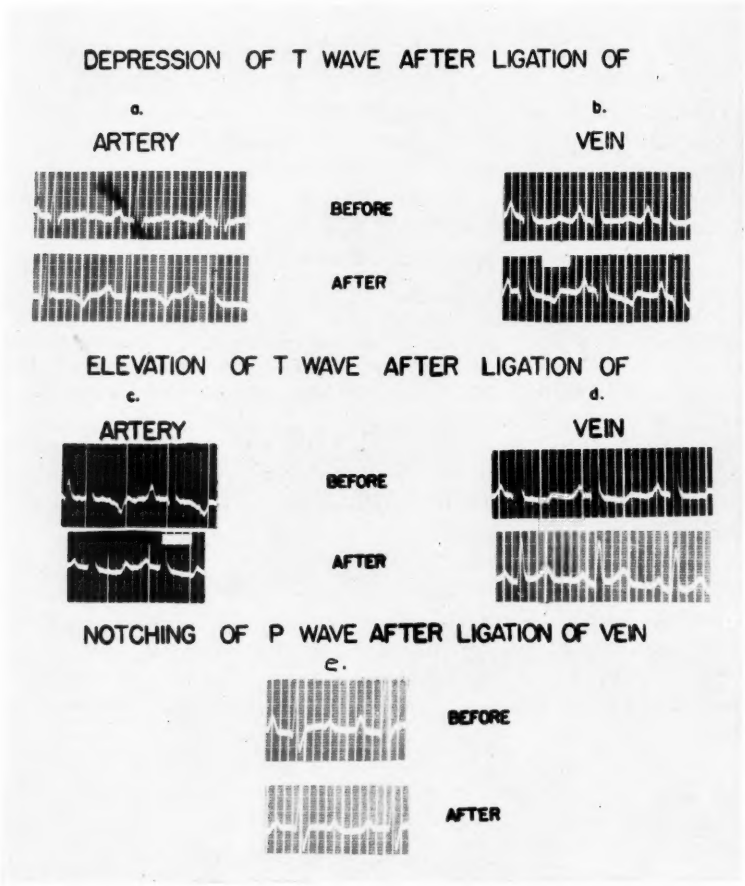

Fig. 1.

Scherf and Schönbrunner[5] have suggested that such changes are indicative of spasm of the coronary arteries, conclusive data in this regard are lacking. Nevertheless, the changes in T wave are important in that they prove that manipulation of the hilar vessels influences cardiac action. It is unlikely that these changes were due to generalized anoxemia in the present study; they occurred only as the pulmonary blood vessels

were being manipulated. The lung to which these vessels were attached was collapsed early in the operation and the subsequent interference with its blood supply could not induce generalized anoxemia. It appears, therefore, that the electrocardiographic changes observed were reflex in origin, as has been suggested by other workers.[5, 7] It should be noted that in addition to electrocardiographic changes, other abnormalities of cardiovascular function have been shown to be induced by reflexes originating in the lungs.[8, 9]

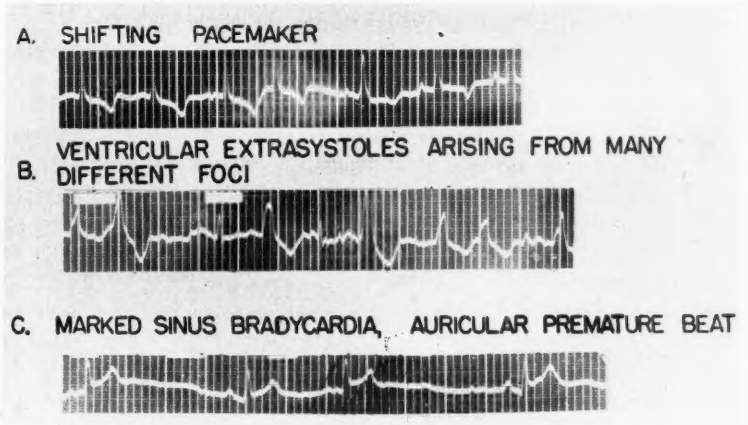
A. SHIFTING PACEMAKER
B. VENTRICULAR EXTRASYSTOLES ARISING FROM MANY DIFFERENT FOCI
C. MARKED SINUS BRADYCARDIA, AURICULAR PREMATURE BEAT

Fig. 2.

In the present small series of experiments, five instances of transient cardiac arrhythmia occurred. These included one case of marked cardiac slowing and one of numerous ventricular extrasystoles arising from different foci, almost entirely replacing the normal beats; the latter is considered a precursor of ventricular fibrillation. A number of authors[3, 4, 10, 11] have described auricular flutter or fibrillation, ventricular tachycardia or fibrillation, and various degrees of heart block after clamping the main pulmonary artery or during fatal pulmonary embolism in animals. The occurrence of these arrhythmias as a result of these drastic procedures is of interest, but is probably not significant in regard to the problem of sudden death during operations on the lung in man. The disturbance of cardiac dynamics resulting from complete or almost complete obstruction to the outflow of blood from the right ventricle occurring during the studies described by these workers[3, 4, 10, 11] is much more marked than that occurring during lobectomy or pneumonectomy.

The results of the present experiments suggest that operations on the lung in man may influence cardiac action, probably through reflexes originating in the hilus of the lung. The observation that numerous ventricular extrasystoles arising from many different foci and marked

bradycardia sometimes occur during experimental pneumonectomy, suggests that the sudden deaths occasionally occurring in man during operations on the lung may result from ventricular fibrillation or cardiac standstill.

SUMMARY AND CONCLUSIONS

1. Electrocardiographic studies were made in eleven dogs in which pneumonectomy was performed under pentobarbital anesthesia. These experiments were performed in order to gain information relative to the problem of sudden death during operations on the lung in man.

2. Changes in the T wave of the electrocardiogram occurred in eight experiments. These changes occurred during manipulation of a large branch of the pulmonary artery and, with lesser frequency, during manipulation of a large pulmonary vein.

3. In five instances transient cardiac arrhythmias occurred. Two of these arrhythmias, namely, numerous ventricular extrasystoles arising from many different foci and very marked bradycardia, might have proved fatal if more prolonged.

4. The changes in electrocardiogram observed are interpreted as probably due to the action of reflexes originating as a result of trauma to the hilus of the lung.

REFERENCES

1. Otto, H. L.: The Effect of a Sudden Increase in the Intracardiac Pressure Upon the Form of the T Wave of the Electrocardiogram, J. Lab. & Clin. Med. **14:** 643, 1929.
2. Anderson, J. P.: Electrocardiographic Findings in Experimental Pulmonary Embolism, Am. Heart J. **9:** 104, 1933.
3. Krumbhaar, E. B.: Note on Electrocardiographic Changes Accompanying Acutely Increased Pressure Following Pulmonary Artery Ligature, Am. J. M. Sc. **187:** 792, 1934.
4. McGinn, S., and White, P. D.: Acute Cor Pulmonale Resulting From Pulmonary Embolism: Its Clinical Recognition, J. A. M. A. **104:** 1473, 1935.
5. Scherf, D., and Schönbrunner, E.: Ueber den pulmocoronaren Reflex bei Lungenembolien, Klin. Wchnschr. **16:** 340, 1937.
6. Walder, R.: Elektrokardiographische und histologische Untersuchungen des Herzens bei experimenteller Luft- und Fettembolie, sowie bei Embolie durch Stärkesuspension, Beitr. z. path. Anat. u. z. allg. Path. **102:** 485, 1939.
7. Horn, H., Dack, S., and Friedberg, C. K.: Cardiac Sequelae of Embolism of the Pulmonary Artery, Arch. Int. Med. **64:** 296, 1939.
8. Churchill, E. D., and Cope, O.: The Rapid Shallow Breathing Resulting From Pulmonary Congestion and Edema, J. Exper. Med. **49:** 531, 1929.
9. Schwiegk, H.: Der Lungenentlastungsreflex, Arch. f. d. ges. Physiol. **236:** 206, 1935.
10. Frommel, E.: Les troubles du rhythme cardiaque au cours de l'embolie pulmonaire mortelle: Étude electrocardiographique expérimentale, J. de physiol. et de path. gén. **26:** 247, 1928.
11. Criep, L. H.: Electrocardiographic Studies of the Effect of Anaphylaxis on the Cardiac Mechanism, Arch. Int. Med. **48:** 1098, 1931.

BRONCHOGRAPHY

The Use of a Modified Intranasal Method and a Movable Table.

B. A. Dormer, M.D., D.P.H., T.D.D., J. Friedlander, M.B., Ch.B., D.P.H., and M. Gibson, Grad. I.E.E.
Durban, Union of South Africa

BRONCHOGRAPHY (or pneumonography), which is the study of x-ray pictures of the chest after the introduction of contrast media into the tracheobronchial tree, was first begun by Chevalier Jackson[5] in 1918 by means of the bronchoscope. He instilled dry bismuth subcarbonate powder into the tracheobronchial tree as an aid to x-ray diagnosis of the chest. Since then various radiopaque oils, prominent among which is the French preparation lipiodol,[11] have been used for the same purpose. Forestier[11] in 1922 showed the first bronchial pictures made after lipiodol injection. Bronchography has, since its introduction, greatly increased our knowledge of bronchopulmonary lesions.

Various methods for instilling the radiopaque oil into the bronchi have been evolved, and the following are the methods in use:

(a) Laryngeal method or supraglottic injection.
(b) Transglottic method (true intratracheal method).
(c) Subglottic (or transcutaneous) method.
(d) Intrabronchial method (bronchoscopy).
(e) Nasal method.

(a) *The Laryngeal Method (or Supraglottic Injection).*—In this method the oil is dropped into the vestibule of the larynx by means of a short cannula bent at an angle; the patient is seated, and his tongue is drawn out of his mouth as far as possible. The cannula, attached to the syringe filled with lipiodol, is carried back to the epiglottis. The patient is told to breathe deeply, and with each inhalation the oil is injected drop by drop. The velum and the base of the epiglottis are sometimes anesthetized, and the oil is injected under guidance of the laryngeal mirror. The oil will then gravitate into the bronchial tree. The disadvantages of this method are that very often some of the oil passes into the stomach and, as patients are in the sitting position, only the bronchi at the lung bases can be delineated.*

(b) *Transglottic (or True Intratracheal) Route.*—This method, first devised by Forestier and Leroux, consists of dropping lipiodol directly into the trachea by means of a long cannula introduced between the vocal cords. After the velum, the epiglottis and the laryngeal cavity are anesthetized, the specially curved cannula (e.g., Rosenthal's cannula) is passed between the cords with the aid of a laryngeal mirror. The patient breathes quietly, and the oil is slowly injected

From the King George V Jubilee Hospital for Tuberculosis, Durban.
Received for publication, July 7, 1941.

*By quickly changing the patient to a "head-down" position lying on the side to be studied, we have found it possible to visualize even the upper lobe bronchus in many hundreds of cases by this simple method which was first described by J. J. Singer. *Editor.*

into the trachea. This method has the distinct advantage in that the operator knows the oil is passing into the trachea. On the other hand, a certain amount of skill is required in passing the cannula between the vocal cords.

(c) *Subglottic Method (Transcutaneous or Cricothyroid Method).*—In this method a needle is passed directly into the lumen of the trachea through the cricothyroid membrane. The patient is placed on a couch in the x-ray room with the shoulders somewhat elevated and a sandbag behind the neck so as to keep the neck well extended. The overlying skin having been anesthetized, a firm needle is passed at right angles through the cricothyroid membrane, and 5 per cent cocaine solution is quickly injected into the trachea; otherwise the patient may cough before the needle is withdrawn. The special lipiodol needle is then inserted through the cricothyroid membrane and the lipiodol is slowly injected. The patient is then turned into whichever position it is considered necessary for the oil to gravitate.

This method has very definite disadvantages. Patients as a rule dislike the idea of needles being inserted into the trachea. False passages may occur; the needle may be ejected from the lumen of the trachea during a fit of coughing and the lipiodol may be injected into the pretracheal space and trickle down into the mediastinum. Subcutaneous emphysema may occur, and the needle track may become infected.

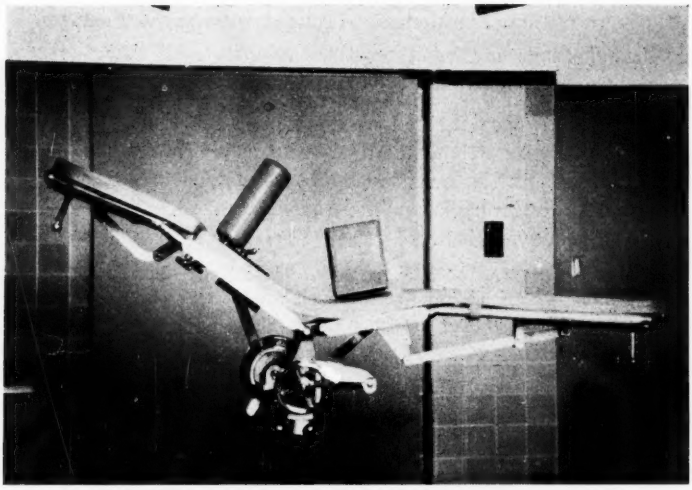

Fig. 1.—The table used for bronchography.

(d) *Intrabronchial Route (Bronchoscopic Method).*—This method was first employed by D. H. Ballon, of Montreal, Canada. He used the bronchoscope to inject lipiodol directly into the primary bronchi and even into pathologic lung cavities. The technique is that used in ordinary bronchoscopy. In skilled hands this method is valuable, as it permits of preliminary aspiration of the secretions and the injection of the particular area to be studied. For this method the operator must be a skilled bronchoscopist and the patient must be in a fairly good condition to stand the procedure of bronchoscopy.

(e) *Nasal Method.*—The patient sits upright, and one side of the nose is sprayed with cocaine solution. Either side of the nose may be used provided there is no

obstruction from a deflected septum. The throat is then anesthetized by means of a de Vilbiss spray. After five to ten minutes, a gum elastic catheter is introduced into the nose until it reaches the posterior pharyngeal wall, when it will turn downward. The head of the patient is now bent slightly forward. The patient is told to breathe regularly and deeply, and the catheter is gently pushed on during inspiration, when it will be found to glide quite easily into the larynx. It may now be pushed down the trachea. Usually the patient coughs slightly as the catheter passes into the larynx. The larngeal mirror may be used to make certain that the catheter is in the larynx. Some cocaine solution is injected via the catheter to anesthetize the mucous membrane of the trachea.

(f) *The Modified Intranasal Method.*—Ellman[3] in 1939 described a method of introducing an iodized oil into the tracheobronchial tree through the nose without the use of any intranasal catheter. The method in use at the King George V Hospital for Tuberculosis, Durban, is largely based on Ellman's method.

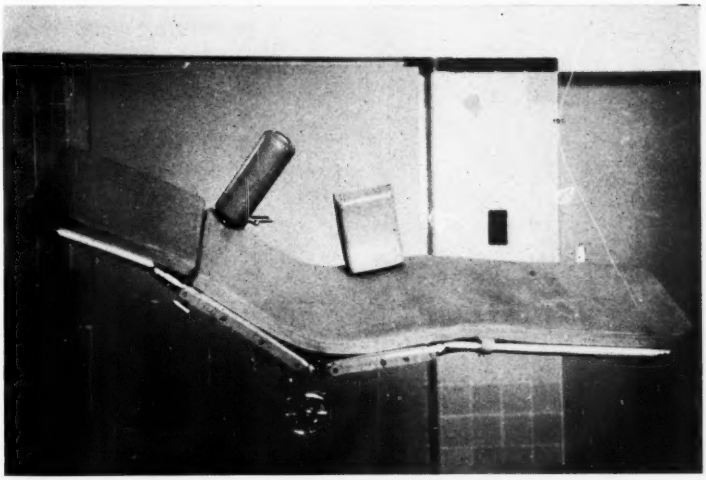

Fig. 2.—The table with a slight lateral tilt.

Before lipiodol is injected, the patient is tested for any sensitivity to cocaine and iodine. Cocaine (gr. 1/10) is injected hypodermically, and the pulse is taken every fifteen minutes for an hour. If there are no appreciable increase in pulse and no complaint of faintness, we assume the patient is not sensitive to cocaine. To test any sensitivity to iodine, the patient is given potassium iodide (gr. 5 to 10) in a mixture of ½ oz. t.d.s. for three days. If there are no signs of "iodism" after that period, it is assumed there will be no untoward effects from the iodine of the lipiodol. (In patients showing sensitivity to iodine, it is unwise to instill lipiodol. In such cases a bromine containing oil [bromipin] may be used if there is no sensitiveness to bromides.)

Before bronchography is commenced each stage of the procedure is carefully explained to the patient in order to gain his confidence and cooperation.

In the case of young children we have obtained excellent results by giving a hypodermic injection of 1/12 to 1/6 gr. of omnopon about one-half hour before the operation. The child loses all appearances of anxiety and is able to cooperate quite easily.

The patient is given ½ tablet "decicain" gr. 1½ (syn pantocain) to suck, or he is told to gargle with a pleasant-tasting gargle in which ½ tablet of decicain is dissolved. As a result of the anesthetizing effect of this on the pharynx and nasopharynx, we find we need use very little cocaine solution. A 2.5 per cent cocaine hydrochloride solution is used for spraying the larynx and the nose. For a couch we use the Brompton Hospital Model Operating Table (Genito-Urinary Company), which is capable of being moved into any position required.

The table is placed in the x-ray room, and a picture of the patient in the recumbent position may be taken as soon after the injection of the lipiodol as desired. For the actual injection, we use a Roberts lipiodol syringe, a short bit of rubber tube, and a nasal olive. The patient's tongue is held well out, and the patient is advised to breathe quietly through the mouth and on no account to swallow or cough. The lipiodol, previously warmed, is then slowly injected via the nasal olive which is inserted into one nostril while the other is held closed. The lipiodol makes a gurgling noise as it trickles down the trachea. As a rule, we use only 10 c.c. of lipiodol for each lung, though more may quite safely be given.

Fig. 3.—The intranasal method of introducing lipiodol into the tracheobronchial tree.

Before the lipiodol is introduced, a plain x-ray picture of the chest is taken to localize the lesion in the lung field.

With the aid of the movable operating table, the patient may be placed in any position from an extreme Trendelenburg to a sitting position, and tilted far over to the left or over to the right. It is important to visualize the direction and anatomical position of the bronchi so that the patient may be placed in the best position for allowing the lipiodol to reach the site of the lesion.

THE PATIENT'S POSITION

Lesion at Base.—The patient is moved up into the Fowler's position, with the head slightly back and the table tilted to the particular side.

We find that the sitting position with the torso erect is not too satisfactory as the oil tends to be held up at the carina and spill over to the opposite side. If it is desired to do both bases together for comparison, we prefer to do one base first, then the other at the same sitting by first introducing the lipiodol to the one base and then tilting the table and introducing the oil into the other.

Lesion at Mid-zone.—The patient is placed in a lesser Fowler's position and somewhat more tilted to the particular side than in the above position.

Lesion at Apex.—To place the oil in the region of the apex, the patient is tilted to the extreme position of that side and lies with a slight downward tilt so as to allow the oil to run as far as the carina. As soon as it is judged that the lipiodol has reached the carina (about two to three minutes), the patient is turned into the extreme Trendelenburg position, but still maintaining the tilt to the particular side. In this way the oil may pass along the ascending bronchi to the apex.

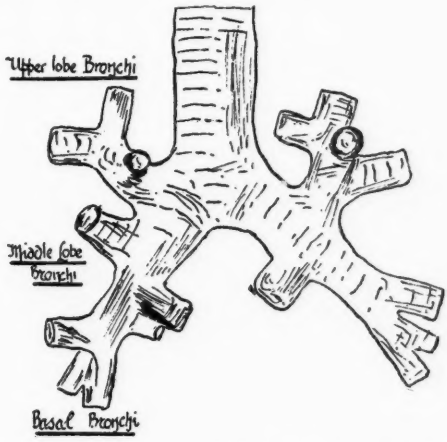

Fig. 4.—Diagrammatic representation of tracheobronchial tree.

After several minutes to allow the lipiodol to reach the terminal bronchi, the patient is slowly returned to the horizontal position; an x-ray picture is then taken in the recumbent position. For a basal lesion the roentgenogram is taken with the patient erect; for all other lesions roentgenograms are taken with the patient in the horizontal position. As a routine we always take a lateral as well as an anteroposterior picture.

RADIOGRAPHIC CONSIDERATIONS

The prime factors for a good bronchograph are: (1) high speed, (2) high power, and (3) high penetration. (1) Limits all possible move-

Fig. 5.—Lipiodol in a lesion at apex. This is the same patient as in Fig. 7. The oil was first introduced into the left base (as in Fig. 7) and later (at the same sitting) into the right apex.

Fig. 6.—Lipiodol in a lesion at mid-zone.

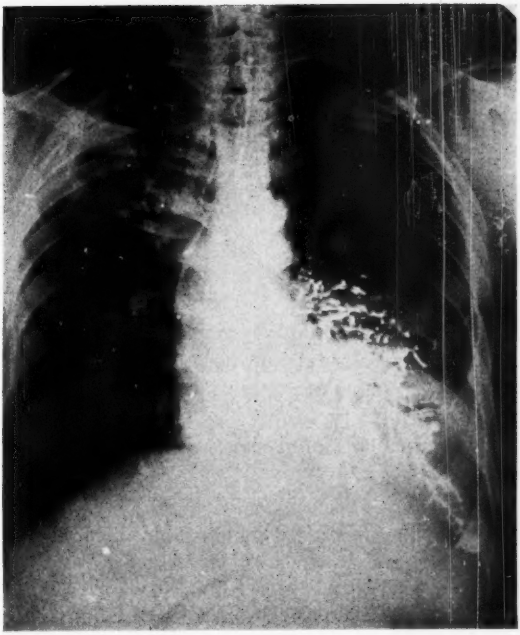

Fig. 7.—Lipiodol in a lesion at the left base.

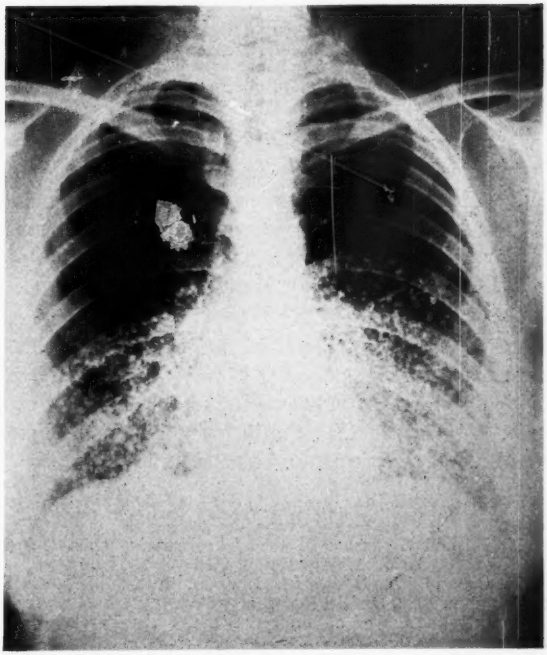

Fig. 8.—Normal bronchogram. Ten cubic centimeters of lipiodol were introduced into the one base and, by tilting the table to the opposite side, 10 c.c. were introduced into the other base.

ment; (2) is necessary in view of the short exposure time; and (3) is necessary in order to show fine bronchi filled with lipiodol through the heart, mediastinum and through the shadow of the diaphragm. The apparatus used is a Philips chest plant where a condenser discharge of 0.03 second is employed with a rotating anode tube. The milliamperage may be preset at 250 to 500, and the peak kilovoltage is adjustable up to about 85. In the horizontal ray position, the patient stands on an electric lift, the tube and casette holder being at a fixed height. The tube distance is variable from 110 to 150 cm., and it may be wound back to the vertical ray position where a distance of about 110 cm. is used; this can be changed by raising the table. Screening facilities are available on this plant which are exceptionally useful where an oblique is required in the upright position. The screening tube is brought in front of the rotating anode, and the screen itself is in front of the casette holder.

The reproductions have been made by means of a Contax III Camera with a Sonar F/1.5 lens on a panatomic film. The miniature photographs are then enlarged up to a convenient size. This method produces results superior to the ordinary reducing camera.

COMMENTS

The method as described is very simple and safe. It has on occasion been employed by only one operator without assistance. The preliminary testing with iodine and cocaine practically precludes the danger of any idiosyncrasy to these drugs. The actual instillation of the oil causes no discomfort and does not upset the patient. We have employed this method in young children without any difficulty and have on occasion used it in very ill patients in whom a diagnosis was essential without any undue distress. In our series of over 250 cases done by this method, we have invariably had good results, and no patient has shown any untoward reaction. By using a preliminary injection of omnopon in young children, we have obtained good results without the use of general anesthesia.

By means of the movable table (with its shoulder and side supports) we are able to place the oil at the site of the lesion after having worked out the direction of flow from a knowledge of the anatomy of the tracheobronchial tree. In view of this, the introduction of the lipiodol under the guidance of a fluoroscopic screen becomes unnecessary.

REFERENCES

1. Arbuckle, M. F., and Stutsman, A. C.: The Bronchoscopist and the Thoracic Surgery Team, J. A. M. A. **113**: October, 1939.
2. Chandler, F. C., and Wood, W. B.: Lipiodol in the Diagnosis of Thoracic Disease, London, 1928, Oxford University Press.
3. Ellman, P.: Introduction of an Iodised Oil Into the Tracheo-Bronchial Tree, Lancet **1**: February, 1939.
4. Gilroy, Glass, E. J.: Bronchography, Brit. M. J., No. 4114, November, 1939.

5. Jackson, C.: The Bronchial Tree: Its Study by Insufflation of Opaque Substances in the Living, Am. J. Roentgenol. **5**: October, 1918.
6. Jewesbury, E.: Bronchography Without Cocaine, Brit. M. J., No. 4084, April, 1939.
7. McGuckin, F.: A Method of Injection of the Bronchial Tree With Lipiodol, Newcastle M. J., January, 1933.
8. Neil, H. T., Gilmour, W., and Gwynne, F. J.: The Anatomy of the Bronchial Tree, Brit. M. J., No. 4079, March, 1939.
9. Nelson: Diagnostic Roentgenology.
10. Rudman, E.: Bronchial Catheterisation, Am. Rev. Tuberc. **39**: March, 1939.
11. Sicard and Forestier: The Use of Lipiodol in Diagnosis and Treatment, Milford, 1932, Oxford University Press.

ASPIRATION OF THE LOWER AIR PASSAGES FOLLOWING ENDOTRACHEAL ANESTHESIA

CLARENCE E. BIRD, M.D.
PROVIDENCE, R. I.

ALTHOUGH much has been written on the subject of postoperative atelectasis and pneumonia, no one until Meusel (1940),[10] appears to have focused attention on the importance of emptying the bronchi completely at the close of operation. The papers by Faulkner (1930, 1932),[4, 5] Lubin (1933),[9] Haight (1938),[7] and Beecher (1940),[2] gave this matter insufficient consideration, in my opinion.

Two years ago, one of my patients died on the third day following an esophagectomy for carcinoma (Case 4). While my impression had been that death was due to fulminating mediastinitis, the autopsy showed simply a purulent bronchitis, accompanied by atelectasis of the left lower lobe and patchy lower lobe pneumonia. Since this unpleasant experience, our anesthetists and I have been very much on the alert to keep the pharynx and large air channels as free of secretion as possible at all times. Also, we have learned to be especially sure that the trachea and large bronchi are clean and dry before the patient leaves the operating room.

RATIONALE OF BRONCHOSCOPIC ASPIRATION (UNDER DIRECT VISION) IMMEDIATELY FOLLOWING OPERATION

Ordinarily, during an operation, unless mucus, pus, or blood is passed into the bronchial tree in excessive amounts, it may be removed fairly satisfactorily by means of a catheter passed through the endotracheal tube. However, at the close of operation, immediately after such a blind aspiration and before dressings are applied, if one listens to the lungs with a stethoscope, he will often hear bizarre, coarse râles over various portions of the bronchial tree. If he removes the endotracheal tube, taking care to aspirate below the tube as it is withdrawn, then listens again, he will usually still hear some of these râles, arising from fluid in the large bronchi. If, now, a bronchoscope is introduced, exudate is always found, usually in small amounts distributed irregularly along the sides of the trachea and large bronchi and in the orifices of some of the secondary bronchi. After removing this bubbling fluid, on auscultation, the lungs will be found entirely clear.

I believe that the removal of this residual, even though small, is of the greatest importance. If it remains, it may cause localized bronchial

Read (by title) before the Twenty-Fourth Annual Meeting of the American Association for Thoracic Surgery at Toronto, Canada, June 9, 10, and 11, 1941.

obstruction and an exhausting, ineffective cough. This cough, the dangers of which have been emphasized by Brown and Archibald,[3] and Beecher,[1] is ineffective for obvious reasons, such as pain, narcosis, weakness, inefficient posture, decreased vital capacity, and paradoxical movements of the chest wall, diaphragm, heart, and great vessels. If the secretion which the patient is attempting reflexly to expel is heavy and tenacious, it may, fortunately, come up. If it is thinner and more widely distributed, it is more likely to descend deep into the bronchial tree, and may even fill alveoli with pus. Material which might easily have been removed by the bronchoscope before the recovery of the cough reflex, thus may do incalculable harm by being allowed to remain.

Those who read the case reports will note that in two instances of total pneumonectomy (Cases 7 and 19), I found, on inspection immediately after operation, that secretion filled the stump of the main bronchus (Fig. 1). At later bronchoscopic examinations on these patients, this dead space was always found to contain mucus or pus. In both

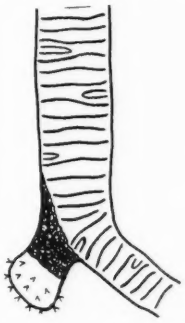

Fig. 1.—Diagram to show the puddling of secretions in the stump of the main bronchus following a pneumonectomy; this infected material should be removed under direct vision immediately after operation, and sometimes repeatedly during the early postoperative course.

patients, whenever they were turned on the side of the sound lung during early convalescence, a fit of coughing would occur, presumably because some of the material spilled over by gravity, as described by Faulkner,[4] into the air stream of the opposite main bronchus. This collection in the stump of the bronchus should be removed whenever it accumulates, but especially should it be aspirated immediately postoperatively, before changes in the position of the patient are made.

Everyone agrees that at the end of an operation the lobes should be re-expanded or left in such a state that they will inflate.* If secretion is puddled in a lobar orifice, adjacent to the stump of a lobe which has

*To effect the re-expansion of a lobe under direct vision before closure of the thorax, carefully controlled positive pressure might conceivably be applied directly to a lobar orifice through a bronchoscope. It takes several times as much positive pressure to re-expand a lobe as to keep it expanded. In other words, it might be preferable to apply directly to the main bronchial orifice the minimal pressure necessary to expand the lobe rather than to subject all lobes, unilaterally and bilaterally, to an equal pressure.

been removed, the tributary segment of lung may remain airless in spite of hyperventilation* and negative pressure applied in the pleural cavity. It is impossible to be sure that the bronchi leading to atelectatic areas are free of secretion unless they are observed directly, and it is my impression that aspiration immediately postoperatively offers the best insurance against the common failure of lobes to re-expand promptly. Furthermore, unless the bronchi are known to be clear, neither the expansion of lobes by positive or negative pressure nor the use of hyperventilation seems safe to me, since it appears probable that the deepening of respiratory excursions at times when the bronchi contain secretions has resulted sometimes in atelectasis and pneumonia, due to a firmer and deeper fixation of secretions in the main air channels or even in the alveoli.

Under endotracheal anesthesia, if a high partial pressure of oxygen is maintained, the tissues may remain quite well oxygenated even though only a small amount of lung is functioning. Lobes and parts of lobes may be atelectatic. The sudden withdrawal of such a mixture should, of course, always be avoided because this is particularly hazardous and may even prove fatal if it occurs when the lungs are nearly collapsed. Direct examination of the bronchial tree, followed by hyperventilation, using a gradually decreasing proportion of oxygen and substituting room air, obviates this danger by insuring the re-expansion of at least a large part of the remaining pulmonary tissue.

To summarize, and amplify, the advantages of immediate postoperative bronchoscopic aspiration, completed before the recovery of the cough reflex, are as follows:

1. The procedure prevents the sucking or driving, by hyperventilation or ineffective cough, of foreign material into lobes and lobules.

2. It favors the re-expansion of collapsed segments of lung, when positive pressure is applied by mask and negative pressure is exerted within the pleural space.

3. It allows patients to lie flat and on the side, instead of in the Trendelenburg position (Gray[6]), in this way conserving vital capacity.

4. It minimizes exhausting coughing, thus lessening circulatory strain and allowing the patient more complete rest.

The possible disadvantages are that:

1. On rare occasions the anesthesia might have to be deepened in order to carry out bronchoscopy.

2. In inept hands, bronchoscopy may be damaging in itself.

Up to the present time we have not been obliged to deepen the anesthesia in order to perform a satisfactory bronchoscopic aspiration.

*Hyperventilation should be carried out with carbon dioxide which is mixed with air, nitrogen, helium or another gas which is not readily absorbed into the blood stream, never with oxygen or cyclopropane which are readily absorbed and might tend to favor or maintain atelectasis.

These aspirations are carried out by the bronchoscopists of the hospital, the anesthetists, or the surgeons, who are trained to do this easily, rapidly, and without trauma to the larynx, trachea, or bronchial mucosa. I have seen upper incisor teeth loosened on two or three occasions, but otherwise there has been no damage. I do not think that skillfully performed bronchoscopy, done with the aid of a laryngoscope and with the head held properly, harms the patient significantly.

PROCEDURE

Our procedure in thoracic operations is as follows: In the wet cases, effective postural drainage is carried out two hours before the anesthetic is given, as recommended by Haight,[7] Beecher,[1, 2] and others. In all except very dry cases, bronchoscopic aspiration is done under local anesthesia immediately before the introduction of the endotracheal tube. During the operation, aspiration is repeated

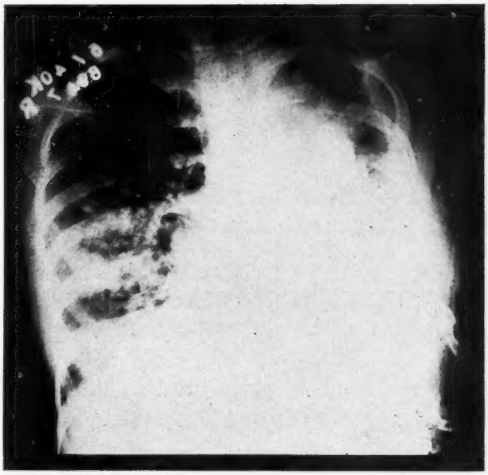

Fig. 2.—Extensive bronchopneumonia in the right lung, five days after a left total pneumonectomy (Case 7); two days later, a tracheotomy tube was introduced to facilitate the frequent aspiration of large amounts of mucopus from the right bronchial tree.

often, using a catheter with an open end and one side hole near the end. (This catheter should not be too soft, so that its direction can be controlled somewhat, and it must be long enough to reach the lowermost branches of the main bronchi. The suction must be efficient and dependable.) At the end of the operation, the pharynx is sucked out and the trachea and bronchi are aspirated through the endotracheal tube. With the catheter still in place, this tube is withdrawn, bringing the suction tip slowly along the trachea, larynx, and pharynx behind it. A bronchoscope is then introduced through a laryngoscope and every portion of the bronchial tree and the trachea and larynx made perfectly clean and dry under direct vision. When it is certain that there is no mucus or pus in the large bronchi, hyperventilation under moderate positive pressure is applied as effectively as possible by mask and at the same time controlled negative pressure is exerted in the pleural cavity, expanding all lobes with air to the desired degree.

Bronchoscopic aspiration at the close of operation does not, of course, prevent all subsequent respiratory difficulties, but in every case it gives the patient the best possible chance of avoiding them, the best possible "send-off," so to speak. Our practice is to use nasal oxygen instead of a tent, in order that frequent and complete changes of position can be carried out (patient on back, on left side, on right

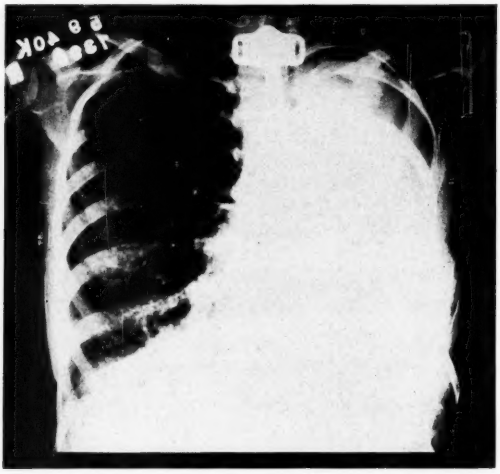

Fig. 3 (Case 7).—Improvement following six days of bronchial aspirations by way of the tracheotomy tube.

Fig. 4.—Equipment for aspirating the bronchial tree through a tracheotomy tube, using a sterile technique.

side, then almost over on the abdomen with the foot of the bed slightly elevated). We aspirate the pharynx frequently. We are continually on the alert for reaccumulation of moisture, and if the patient does not quickly and completely empty the bronchial tree by coughing, we do not hesitate to do a bronchoscopic examination in the patient's room. This is done simply and rapidly, but with no neglect of essentials, such as adequate assistance, well-cared-for sterile instruments, and

efficient suction. If the bedstead is in the way, the mattress is swung part way around on top of a bedside table. When the patient is pulled out over the end of the mattress, he is in good position for bronchoscopy. Larocaine anesthesia may be used, or none at all. We have not used transnasal aspiration, as described by Haight,[7] because all necessary equipment has been available and we consider that bronchoscopic aspiration is more certain and thorough.

Jackson and Jackson[8] have said that "pneumonia after tracheotomy is one of the curiosities of medicine." Similarly, it has been my experience following laryngectomies (Cases 1, 8, 12, and 22), that although repeated aspirations through the tube are necessary, pulmonary complications rarely occur. Tracheotomy seemed life-saving in one girl (Case 7) in whom the right lung became extensively involved in a bronchopneumonic process (Fig. 2), a few days following the removal of the left lung for bronchiectasis. The patient was too weak to cough effectively and large amounts of thick, purulent material were aspirated by catheter from the bronchi over a period of two weeks before the tracheotomy tube could be safely removed (Fig. 3). No doubt sulfathiazole contributed also to her recovery.

The sterile setup for aspirating through a tracheotomic cannula is shown in Fig. 4. A freshly-boiled catheter is used, even if aspiration is necessary as often as every fifteen minutes. The catheters are boiled on an electric plate in the room and two of them, with glass connectors attached, are kept on a sterile sheet with a basin of saline solution and two Kelly clamps alongside. It is necessary for an assistant to support the heavy-walled, noncollapsible suction tubing from above so that the catheter may be manipulated freely. By using care, the procedure introduces no extraneous organisms, since the catheter is handled entirely by the two sterile Kellys.

DISCUSSION

The anesthetists with whom I have been associated have preferred the Waters endotracheal tube with the cuff inflated. I think I should be as well, or better, satisfied with an endotracheal tube which fits loosely, combined with a snug face mask, as recommended by Beecher.[1, 2] However, the head-down position (incline of 20 degrees, patient on side), used by him, lessens the capacity of the lungs on the "down" side and somewhat diminishes the space in which to work on the "up" side. This is due to the weight of the abdominal organs, elevating, as they do, both leaves of the diaphragm. In the wetter cases, it is my impression that this gravity drainage, during operation, is of distinct advantage. However, small amounts of secretion are not much affected by gravity and at the close of the procedure they adhere to the bronchi or alongside the trachea. Therefore, no matter which technique is used, I believe that in cases in which there has been even a minimal amount of secretion in the airways, a bronchoscopic aspiration should be done immediately after operation, before the patient recovers the cough reflex.

If, in thoracic and abdominal cases, a mask is used without an endotracheal tube, pharyngeal contents usually enter the bronchial tree, as shown by Myerson[11] twenty years ago, and, of course, in many thoracic operations secretions enter from the diseased lung. Therefore, it seems evident that bronchoscopic aspiration should also be carried out im-

mediately after operation whenever the mask technique without an endotracheal tube is used.

I think, too, that the bronchoscope should be utilized immediately after operation following many procedures on parts of the body other than the thorax, whenever the stethoscope shows at the close of operation that there is moisture in the tracheobronchial system. This is especially the case in those individuals who for one reason or another are most liable to postoperative pulmonary complications. Included are patients who are short and thick-set, the old and the very young, the ones who have had particularly long operations or difficult anesthesias, and those who may, under some circumstances, have inhaled blood or pus, pieces of tissue, or other foreign material. The point applies particularly to patients who have had upper abdominal operations, whose diaphragms or muscles of respiration are weak or paralyzed, or who for any reason are not likely to be able to cough or respire freely.*

The cases on which the above impressions are based are reported at the end of the article. The thoracic cases are personal and consecutive, dating from Jan. 1, 1939. Only a few particularly interesting laryngectomy and abdominal cases are included. There have been no deaths in any of my personal abdominal cases in which endotracheal anesthesia has been used.

SUMMARY

At the end of long and difficult operations, especially those on the thorax, the region of the diaphragm, and the upper abdomen, there is always some secretion in the bronchial tree which should be removed. This can be accomplished with certainty only by aspiration under direct vision through a bronchoscope.

The advantages of such an aspiration, when carried out while the patient is still anesthetized and before the recovery of the cough reflex, are as follows: (1) the procedure prevents the descent of exudate into lobes and lobules; (2) it favors the re-expansion of collapsed lobes when differential pressures are applied; (3) it allows patients to lie horizontally instead of in the head-down position, in this way conserving vital capacity during the hazardous early postoperative hours; and, (4) it eliminates coughing, thus lessening circulatory strain and allowing more complete rest.

Whenever secretion reappears in the bronchial tree after operation, it should be removed. Unless it is coughed up quickly and completely, bronchoscopic aspiration should be carried out in the patient's room.

In a few cases, if secretions reaccumulate rapidly, tracheotomy is indicated in order that aspirations may be repeated at short intervals with little disturbance.

*It should be mentioned that in all patients, whether or not bronchoscopic aspiration is done, it is most important to empty the pharynx by suction immediately after operation.

LIBRARY
BOSTON UNIVERSITY
SCHOOL OF MEDICINE

Reports are presented of the cases which form the basis for the views expressed.

CASE REPORTS

CASE 1.—A woman, aged 60 years, had extensive epidermoid carcinoma of the hypopharynx, larynx, and upper esophagus, grade 1.

March 5, 1939, a Witzel gastrostomy was done and on March 10, 1939, a pharyngo-laryngo-esophagectomy. The anesthesia was endotracheal insufflation with ether. Care was taken not to allow blood from the field of operation to enter the bronchial tree. Postoperatively, the trachea was kept clean by frequent aspirations through the tracheotomy tube. Several crusts of coagulated blood were removed from the trachea with the aid of a bronchoscope on the third postoperative day. The temperature, pulse, and respiratory rates remained normal after the sixth day.

Obstruction of the trachea was caused by a recurrence of the growth, resulting in death eight months postoperatively.

CASE 2.—A male, aged 65 years, had epidermoid carcinoma of the mid-esophagus. On March 13, 1939, a Witzel gastrostomy was performed; March 18, a tooth extraction; March 19, pneumothorax, left; March 21, sectioning of fourth to seventh intercostal bundles and ribs, left; and March 29, 1939, esophagectomy (Torek). The anesthesia used was basal tribromethanol, endotracheal nitrous oxide and oxygen with cuff inflated. The bronchial tree was aspirated blindly by a catheter during, and at the close of, the operation. Death occurred on the fourth postoperative day. Autopsy showed extensive mediastinal, pleural, and pulmonary infection.

CASE 3.—A male, aged 62 years, had carcinoma of the upper lobe of the right lung.

July 19, 1939, anterior exploration of right lung was done, biopsy of rib and ligation of the right pulmonary artery. The anesthesia was endotracheal cyclopropane with cuff inflated. During and at the close of operation the bronchial tree was aspirated blindly. Postoperatively, there was empyema with atelectasis of the lobes on the right. Tracheotomy was resorted to on the eighth postoperative day to facilitate the aspiration of mucus and pus which the patient was unable to expectorate, but the clinical course was unfavorable. A limited autopsy, on the eighteenth postoperative day, showed carcinoma of the right upper lobe with extensive infiltration of the parietal pleura by the tumor.

CASE 4.—A 54-year-old male had epidermoid carcinoma of the mid-esophagus with metastases to the adjacent lymph nodes.

Feb. 6, 1940, he had a Janeway gastrostomy; Feb. 20, pneumothorax, left; Feb. 23, esophagectomy (Torek). Anesthesia was basal tribromethanol and endotracheal cyclopropane with cuff inflated. The tumor was peeled away with difficulty from the left main bronchus. The trachea and main air passages were aspirated blindly during, and at the close of, the operation. Postoperatively, there was moderate dyspnea followed gradually by the accumulation of secretions in the bronchial tree, with cyanosis and exhaustion. Autopsy, on the third postoperative day, showed seropurulent bronchitis, atelectasis of the left lower lobe, and bilateral bronchopneumonia of the lower lobes. There was no mediastinitis or gross infection of the pleura. Tumor tissue was not demonstrated in sections from the wall of the left main bronchus or elsewhere.

CASE 5.—A patient, 22 years old, female, had stenosis of the lower esophagus. At the age of 3 years, this girl had "swallowed a shoe button with a hook on it." The foreign body was not removed until about one year later. Since that time she had

been unable to swallow solid food, and regurgitated and vomited frequently. Innumerable dilatations were carried out in several clinics, but without relief. By the time of admission she was "getting worse and losing strength." Her weight was 64 pounds.

April 12, 1940, a "Finney type" esophagogastrostomy was carried out by an abdominal approach. The anesthesia was procaine field block and endotracheal cyclopropane with cuff inflated. The trachea and bronchi were aspirated blindly at the close of operation. On the first postoperative day, râles were heard over the trachea and large bronchi. A roentgenogram showed mottled density in both lower lobes; however, a bronchoscopic aspiration was not done because the patient cleared her lungs quite effectively by change of position and coughing. All vital signs were normal after the first forty-eight hours. Following this operation she returned home. Although emptying of the esophagus into the stomach was improved, it was still unsatisfactory.

June 1, 1940, the patient returned to the hospital for a Witzel gastrostomy. On June 28, esophagogastrostomy by a left transpleural approach was done. The anesthesia was endotracheal cyclopropane with cuff inflated. The bronchial tree was aspirated blindly several times during the operation, and again at the close. She became exhausted late on the afternoon of the first postoperative day; the lips were slightly cyanotic, the respiratory rate was 36, and the pulse rate 160. The left lung was clear, but the right lung was dull anteriorly, and coarse ronchi were heard over the entire right chest anteriorly and posteriorly. The breath sounds were suppressed over the lower half of the right chest. The heart was apparently in the normal position. A portable roentgenogram showed atelectasis of the right lower lobe. Bronchoscopic aspiration, performed in the patient's room, brought large plugs of mucopus from the middle and right lower lobe bronchi. Recovery was prompt, and the vital signs were normal after the third postoperative day.

Subsequently, she improved progressively. The esophagus remains dilated. There appears to be much chronic inflammatory tissue at the site of the esophagogastrostomy, although a 9 mm. esophagoscope is passed into the stomach without difficulty. The patient volunteers that she now lies down flat when in bed, whereas before her first operation she had to sit up part way so that she would not regurgitate and choke. Her weight, April 15, 1941, was 87 pounds. Her diet is not restricted and she has no symptoms.

CASE 6.—A woman, aged 48 years, was admitted to the hospital with carcinoma of the upper stomach with extension into the lower esophagus and diabetes mellitus of moderate severity.

April 22, 1940, a left transpleural esophagogastrectomy was performed, with anastomosis of the greater curvature tube to the esophagus. The anesthesia used was endotracheal cyclopropane with cuff inflated. The lungs were re-expanded and the bronchi aspirated frequently during the operation. A small amount of secretion was removed by bronchoscopic aspiration following the removal of the endotracheal tube.

On the morning of the second postoperative day, a moderate collection of mucus was removed from the pharynx, trachea, and bronchi by bronchoscope, and sulfanilamide was given. Late the same afternoon the respirations became slow and gasping, no breath sounds could be heard over the left lung, and the heart was found to be displaced against the left chest wall. There were many coarse tracheal and bronchial râles. A portable roentgenogram showed, on the left, only a small amount of aerated lung at the apex. A large mass of thick, viscid mucus was aspirated from the trachea and bronchial tree with much relief. Hypoglycemia was corrected by intravenous dextrose. Thereafter the lungs were fairly clear, except for the left

lower lobe which remained atelectatic. Another bronchoscopic aspiration, on the ninth postoperative day, brought thick mucus from all orifices of the secondary lower lobe bronchi, following which both lungs expanded freely.

The anastomosis was accomplished in this case by pulling the ligatured esophagus through an incision in the upper end of the remaining tube of the stomach. During the three months after discharge from the hospital several dilatations were carried out. The management of the diabetes became very difficult and finally the patient became very weak, was readmitted, and died in three days. Autopsy showed metastases to the mediastinal lymph nodes, liver, lung, and thoracic wall; also, chronic fibrous pleurisy on the left; chronic localized pericarditis; calculi of the pancreatic duct with obstruction of the duct and marked atrophy of the pancreas; acute splenitis; and chronic cystitis with hydro-ureter on the right. Ten centimeters of the tube of stomach lay within the thorax. The rugae of the stomach were well marked. There were no cancerous areas in the stomach or esophageal wall. The scarred ring at the site of the anastomosis was 7 mm. in diameter, and the esophagus above this measured 15 mm.

CASE 7.—A 15-year-old girl showed bronchiectasis throughout the left lung.

April 26, 1940, the dissection technique was used for a left pneumonectomy. The anesthesia was endotracheal cyclopropane with cuff inflated. Blind aspiration of the right bronchial tree was carried out frequently during the operation. The left main bronchus was sutured flat with silk and allowed to retract into the mediastinal tissues. A bronchoscopic aspiration at the close of the operation brought considerable pus from the air channels and from the stump of the left main bronchus.

April 27, 1940, the first postoperative day (9:15 P.M.), coarse bronchial râles were present over the right chest anteriorly. A moderate amount of mucus was aspirated through a bronchoscope in the patient's room. Immediately afterward, the lungs were clear to auscultation.

April 28, 1940, the second postoperative day (11:30 A.M.), bubbling râles were heard over the right chest. Much mucus was removed by bronchoscope from the entire bronchial tree following which no râles were heard, but the breath sounds were harsher over the lower lobe than on the previous day. At 5:30 P.M., the bronchoscopic aspiration was repeated.

April 29, 1940, the third postoperative day (9:45 A.M.), bronchoscopic aspiration was repeated and a large amount of thin, mucoid material was removed. "A puddle collects each time in the stump of the left main bronchus." The patient was much improved. Sulfapyridine was started and later replaced by sulfathiazole.

May 1, 1940, the fifth postoperative day, she was coughing less and was thought to be improved.

May 2, 1940, the sixth postoperative day (8:15 A.M.), "Has pain on inspiration over the right base. Cough is rather weak because of discomfort. Coarse râles are heard at the base anteriorly." Bronchoscopic aspiration brought about 10 c.c. of thick, tenacious, purulent material which had almost completely filled the large bronchi. The respirations improved immediately. However, the bronchial tree filled so fast during the day that it was apparent the patient could not withstand the respiratory embarrassment long. Therefore, a tracheotomy was done, under procaine anesthesia, and large amounts of thick and thin mucoid secretion were aspirated. Stained smears showed Gram-positive cocci in clusters, and Gram-positive diplococci. The aspirations were continued as necessary, at times every fifteen minutes, sometimes at intervals of as long as three hours. Bronchial breathing, and a few fine crepitant râles persisted over the right lower lobe, and the bronchopneumonic process spread to all lobes. The leucocyte count, at this time, was 33,500, with 98 per cent polymorphonuclears.

On May 6, 1940, the tenth postoperative day, roentgenograms showed beginning clearing of all lobes. Thereafter, heavy yellow-green material was aspirated repeatedly and the pneumonia in the cardiohepatic portion of the lower lobe resolved very slowly.

May 16, the twentieth postoperative day, the tracheotomy tube was removed. By May 23, the twenty-seventh postoperative day, only 3 c.c. of sputum were expectorated during the previous twenty-four hours. A sulfathiazole skin reaction, similar to erythema nodosum, appeared on the legs. The patient had had a total of 85 Gm. of sulfathiazole over a period of twenty days.

June 15, 1940, the fiftieth postoperative day, an empyema was drained by rib resection. There was no bronchopleural fistula. September 16 to November 5, she was readmitted and thoracotomy was repeated for improvement of drainage. Jan. 21 to Feb. 1, 1941, during readmission, the sinus was dilated and a rubber tube drain was replaced.

April 15, 1941, a narrow sinus persisted which extended to the apex of the pleura. The patient is in excellent health. There is no fever and no expectoration.

CASE 8.— A male, aged 39 years, had epidermoid carcinoma of the larynx, grade 2.

May 28, 1940, laryngectomy was performed. The anesthesia was basal ether in oil, per rectum; semiclosed nitrous oxide-oxygen by mask; after division of the trachea, endotracheal ether-oxygen by open catheter. No blood or mucus was allowed to enter the trachea during the operation. The bronchi were sucked out by catheter at the close. For several days, large amounts of bloody mucus were aspirated from the pharynx, but very little secretion was obtained from the trachea and bronchi at any time. The highest temperature, 102.8° F. by rectum, was recorded on the evening of the second postoperative day. There were no pulmonary complications. The patient is now well, at work, and his speech is understood perfectly by his wife and close associates, although imperfectly by others (April 15, 1941).

CASE 9.—A 62-year-old man had adenocarcinoma of the stomach with extension into the esophagus.

July 11, 1940, exploration of the upper abdomen was done, followed by a Witzel gastrostomy. An irregular, quite freely movable mass was found in the fundus of the stomach extending upward in the esophagus. There were a few hard, movable lymph nodes near the diaphragm. The liver did not appear to be involved.

July 19, 1940, left transpleural exploration of the esophagus was made. The anesthesia was endotracheal cyclopropane with cuff inflated. The bronchial tree was aspirated by catheter during the operation. The growth was found to extend within the walls of the esophagus as high as the aortic arch and was considered inoperable. Bronchoscopic aspiration was carried out at the close of the procedure.

The lungs remained clear until the third postoperative day, when the entire bronchial tree became filled with thin mucopus. This was removed by bronchoscopic aspiration, but reaccumulated rapidly. Death occurred on the fourth postoperative day. There was no autopsy.

CASE 10.—A girl, aged 5 years, had a bronchiectatic (?? congenital) cyst of the left lower lobe.

On Aug. 19, 1940, the cyst, which was 10 cm. in diameter and contained viscid, glary mucus, was drained dependently by rib resection. Endotracheal cyclopropane anesthesia with cuff inflated was used. Until the cyst could be emptied externally it was necessary to aspirate large amounts of the mucus through the endotracheal tube. At the close of the operation, bronchoscopic suction left the air channels dry and clean. The temperature, pulse, and respiratory rates remained normal after the first twenty-four hours.

Sept. 11, 1940, the left lower lobe was excised by the dissection technique. The anesthesia was endotracheal cyclopropane with cuff inflated. The bronchi were kept free of mucus by catheter aspirations during the operation. "They were aspirated and were dry when the intratracheal tube was removed." Bronchoscopy was not done at the close of this operation, but eighteen hours afterward it was necessary to bronchoscope the patient in her room to remove a large amount of mucus from the main air channels. The examination was done easily and without trauma, and the relief of dyspnea was complete. However, during the day and ensuing night laryngeal obstruction began, and gradually increased until at forty-two hours postoperatively a laryngoscopic examination showed extensive glottic edema and obstruction. Relief was obtained by introducing a short, No. 5 tracheotomy tube through a transverse incision under procaine anesthesia. Thereafter, frequent aspirations were carried out through the tracheotomy tube. Laryngoscopy, four days later, showed no edema, and the tube was removed. Aspirations were continued for one day through the opening in the skin, following which the patient expectorated spontaneously.

The further convalescence was complicated by empyema, but there was no bronchial fistula and the wound soon healed completely.

CASE 11.—A male, aged 69 years, had epidermoid carcinoma of the lower esophagus, grade 3, with metastases to the pericardium, diaphragm, and liver.

Sept. 14, 1940, a left transpleural exploration was done and the tumor found inoperable. Anesthesia used was endotracheal cyclopropane with cuff inflated. The bronchial tree remained dry throughout the operation. A bronchoscopic aspiration was not done. Postoperatively, there was no cough or pulmonary complication of any kind. Sterile bloody fluid was aspirated from the left pleural cavity on three occasions. This patient was discharged from the hospital unimproved and died two months later.

CASE 12.—A 58-year-old male had epidermoid carcinoma of the larynx, grade 2.

Sept. 14, 1940, laryngectomy was performed. The anesthesia used was endotracheal ether insufflation; after division of the trachea, endotracheal nitrous oxide and ether with the cuff inflated. The trachea and bronchial tree were aspirated by catheter at the close of the operation. There was only a small amount of secretion. Postoperatively, the trachea was kept clean by infrequent aspirations and there were no pulmonary complications. This patient died suddenly at home, two months later, of what was thought to be an overdose of pentobarbital.

CASE 13.—A male, aged 56 years, had adenocarcinoma of the stomach with extension into the esophagus; metastases in the diaphragm and liver.

Oct. 9, 1940, left thoracic exploration and biopsy from the diaphragm were made. Endotracheal cyclopropane anesthesia was used, with cuff inflated. After the operation, the trachea and bronchi were aspirated blindly by catheter while the endotracheal tube was being removed. Bronchoscopic aspiration was not done. The patient was troubled by cough, due to subacute bronchitis, for a few days postoperatively. The temperature became normal on the sixth postoperative day, after discontinuing the administration of sulfanilamide. April 1, 1941, the patient was living, at home, but failing.

CASE 14.—A man, aged 63 years, had epidermoid carcinoma of the mid-esophagus, grade 2.

Oct. 15, 1940, an exploration of the liver was made and Witzel gastrostomy done. On October 23, there was an attempted esophagectomy with endotracheal cyclopropane anesthesia (cuff inflated). The growth was torn open while freeing it from the left main bronchus and the patient died on the first postoperative day as a result

of overwhelming sepsis. The bronchial tree was not aspirated postoperatively. Autopsy showed acute pleurisy and mediastinitis. It was interesting that microscopic sections demonstrated no tumor in the wall of the left main bronchus.

CASE 15.—A 54-year-old male had chronic gastric ulcer (malignancy suspected preoperatively).

Oct. 28, 1940, subtotal gastrectomy was done. Anesthesia used was procaine field block with endotracheal cycloprane, cuff inflated. At the close of the operation, the bronchial tree was aspirated blindly by catheter while the endotracheal tube was being withdrawn.

On the second postoperative day there appeared cough, hyperpnea, and slight cyanosis. These symptoms were accompanied by a rise in pulse and respiratory rates, and a fever. Examination showed atelectasis of the right lung and this was confirmed by a roentgenogram. A bronchoscopic aspiration was done in the patient's room, during which "a considerable amount of heavy, mucopurulent material was removed from the trachea and right bronchial tree; scarcely anything was seen on the left." Thereafter, there was rapid improvement and normal convalescence in every way.

CASE 16.—A boy, aged 8 years, had chronic intussusception of the small bowel.

Nov. 10, 1940, resection was done, with endotracheal cyclopropane anesthesia and ether with cuff inflated. A small amount of bronchial secretion was aspirated blindly by catheter before and during the removal of the endotracheal tube.

On the first postoperative day the boy was unable to cough effectively. A few coarse ronchi were heard over the chest bilaterally. At bronchoscopy, the larynx was observed to be somewhat hyperemic and the cords were swollen. The mucous membrane of the trachea was reddened and the mucosa of the right bronchial tree was quite injected. Only a small amount of mucus was present in the right main bronchus. The left bronchial tree was practically normal. Following this bronchoscopic aspiration the ronchi could no longer be heard and the breath sounds were vesicular over all lobes. The irritating cough stopped.

All vital signs were normal from the fifth postoperative day on. The boy was discharged well and has remained so.

CASE 17.—A woman, aged 38 years, had an internal concealed hernia of the stomach and great omentum, protruding through the gastrosplenic ligament and simulating a diaphragmatic hiatus hernia.

Nov. 23, 1940, the hernia was reduced. The anesthesia used was procaine field block; endotracheal cyclopropane, with cuff inflated. Blind aspiration of the bronchial tree during the removal of the endotracheal tube failed to result in the clearing of coarse râles from the lungs. Bronchoscopic aspiration brought 2 or 3 c.c. of thin secretion from the walls of the bronchi, following which the lungs were found to be normal to auscultation.

From the second to the fifth postoperative days the patient expectorated small amounts of very thick yellowish mucus. Auscultation showed evidence of slight bilateral bronchitis. She was discharged well on the nineteenth postoperative day.

CASE 18.—A 31-year-old woman showed degenerating neurofibroma of the sixth right intercostal nerve.

Nov. 27, 1940, the tumor was excised by posterior mediastinotomy. Anesthesia was endotracheal cyclopropane with cuff inflated. The air passages remained dry throughout the operation, so that at the end when the bronchi and trachea were aspirated with a catheter as the endotracheal tube was withdrawn, very little secretion was obtained. Bronchoscopic aspiration was thought to be unnecessary.

The right pleura had been opened during the operation. The right lung remained partially collapsed for ten days, during which time there was slight fever

with moderate perspiration, a productive cough, and a few râles in the right lung. Further convalescence was rapid and the patient has remained well.

CASE 19.—A male, aged 54 years, had chronic tuberculous pneumonitis with extensive pleural calcification and pulmonary abscess on the right.

Jan. 22, 1941, total right pneumonectomy was done using dissection technique. Endotracheal cyclopropane anesthesia with cuff inflated was used. The bronchial tree was aspirated frequently during the operation and the catheter was brought out behind the endotracheal tube as it was withdrawn. On examination with the stethoscope, many noisy sounds were heard over the remaining lung. Bronchoscopic examination showed only a small residual of thin mucoid material in the left bronchial tree, but there were also 1 or 2 c.c. of mucus lying in the stump of the right main bronchus. All of the secretion was removed, and the distal end of the closed bronchus was seen to be flat and tightly sealed. Auscultation now showed the lung to be free from râles.

On the second postoperative day, the patient looked and felt quite well, but the pulse rate rose to 200, and the rhythm was irregular. A roentgenogram showed a marked shift of the heart, trachea, and great vessels to the right, and the diaphragm was high and the left lung overexpanded. An electrocardiogram indicated auricular flutter with some irregularity. The 70 cm. of negative pressure, exerted by the closed-type water-sealed drainage, were released, and 200 cm. of air were injected into the right pleural cavity. Within thirty minutes, the pulse rate dropped to 108, and the rhythm became regular and strong. A roentgenogram now showed only slight shift of the mediastinal structures to the right and an electrocardiogram was normal.

Sulfathiazole was given for several days and the left lung remained entirely clear. However, until the tenth postoperative day, whenever the patient turned on the left side there occurred a fit of coughing, with expectoration, due, apparently, to the dumping of the contents of the stump of the right main bronchus over the carina. There was no bronchopleural fistula at any time.

Following an extension of tuberculosis to the left lung, this patient died in the Charles V. Chapin Hospital (for infectious diseases), two months after operation.

CASE 20.—A woman, aged 56 years, had adenocarcinoma of the stomach with extension into the lower esophagus; metastases to the regional lymph nodes and liver.

Jan. 23, 1941, abdominal exploration was carried out under endotracheal cyclopropane anesthesia with cuff inflated. The bronchi remained dry during the operation and bronchoscopic aspiration was not done. This patient recovered without bronchitis or other pulmonary complication and left the hospital unimproved.

CASE 21.—A girl, 6 years of age, had a sequestration cavity in the middle lobe, the result of a posttonsillectomy abscess which was drained by thoracotomy eight months previously.

April 30, 1941, the middle, and a portion of the right lower, lobes were removed. Anesthesia used was endotracheal cyclopropane with cuff inflated. During the latter part of the operation small amounts of bloodstained, frothy mucus were aspirated blindly from the trachea and bronchial tree. Before recovery of the cough reflex, a bronchoscope was introduced and small collections of the same type of mucus were removed from the trachea and right and left bronchi. All of the bronchial orifices were seen to be free from secretion. Moderate negative pressure was then applied to the catheter in the right pleural cavity, and positive pressure of 6 mm. of water was applied by mask. Breath sounds were clear over the left lung, somewhat distant over the right upper lobe, and barely heard over the right lower lobe. A roentgenogram of the chest taken two hours postoperatively with

the portable apparatus, showed the right upper lobe two-thirds expanded but there was density over the lower right lung field and the heart was displaced slightly toward the right.

In this child there was much difficulty postoperatively from accumulation of mucus in the bronchial tree, so much so that bronchoscopic aspirations were done twice on the first postoperative day, and a tracheotomy tube was inserted on the morning of the second postoperative day. Large amounts of mucus were removed repeatedly by aspiration through the tracheotomy tube and within a few hours the left lung was practically free of râles and the breath sounds were good over the right upper lobe. The tracheotomy tube was left in place for eight days. Recovery was progressive, with gradual re-expansion of the right upper and lower lobes. Cultures taken of the pleural fluid on the second postoperative day showed a few staphylococci, but the fluid was clear red. The pleural drainage tube became occluded on the third postoperative day and was removed. The wound healed solidly and there was no empyema.

The child was discharged from the hospital, May 22, 1941, and has remained well (June 4, 1941).

CASE 22.—A 63-year-old man had epidermoid carcinoma of the larynx, grade 2, extending into the trachea.

May 21, 1941, a laryngectomy was performed, with resection of the trachea to the eighth ring posterolaterally on the right. Vinethene and ether induction through the tracheotomy tube were used; endotracheal cyclopropane with cuff inflated. After isolating the larynx and upper trachea, the trachea was severed between the second and third rings. The flat, papillomatous tumor was found to be sharply demarcated at the level of the sixth tracheal ring, posterolaterally on the right. By suitable undermining of skin flaps and the removal of tissues down to the sternomastoid muscles and sternal notch, it was possible to divide the trachea between the fifth and sixth rings on the left, then attach the skin flap on the left to the sixth ring with silkworm gut sutures, finally resecting the tumor with a 5 mm. margin and attaching the skin all around, anteriorly, posteriorly, and on the right, to the sixth, seventh, and eighth rings. This gave a mucous membrane-to-skin approximation for the entire circumference. The work was done with a cuff in the lower trachea, inflated over the endotracheal tube. The anesthetic was taken well and the trachea and lungs were dry throughout the operation. A small amount of frothy, bloody mucus was aspirated blindly from the bronchi immediately postoperatively. Within one-half hour the patient coughed effectively, removing slightly bloody mucus from time to time.

A few aspirations of the trachea with a catheter were carried out during the first three postoperative days, but by changing the laryngectomy tube every twelve hours and cleaning the inner tube every two hours, combined with coughing and change of position, aspirations were unnecessary thereafter. The patient was allowed up in a wheel chair on the sixth postoperative day. A Levin tube was passed at the time of operation and all feedings and fluid were given through it for fourteen days. There was no leakage from the pharynx and the patient was discharged from the hospital on the seventeenth postoperative day.

REFERENCES

1. Beecher, H. K.: Principles of Anesthesia for Lobectomy and Total Pneumonectomy, Acta med. Scandinav. supp. 90, pp. 146-157, 1938.
2. Beecher, H. K.: II. Some Controversial Matters of Anesthesia for Thoracic Surgery, J. THORACIC SURG. **10**: 202-219, 1940.
3. Brown, A. L., and Archibald, E.: The Action of Cough Upon Material in the Tracheobronchial Tract, Am. Rev. Tubere. **16**: 111-122, 1927.
4. Faulkner, W. B., Jr.: Internal Drainage; Its Application in Pulmonary Suppuration, J. A. M. A. **95**: 1325-1328, 1930.

5. Faulkner, W. B., Jr., and Faulkner, E. C.: Internal Drainage; a Causative Factor in the Production of Postoperative Massive Collapse of the Lung (Pulmonary Atelectasis); Suggestions as to Prevention and Treatment, Acta chir. Scandinav. **69**: 105-119, 1932.
6. Gray, H. K.: Postoperative Pulmonary Complications and Postoperative Use of the Trendelenburg Position, Minnesota Med. **18**: 273-276, 1935.
7. Haight, C.: Intratracheal Suction in the Management of Postoperative Pulmonary Complications, Ann. Surg. **107**: 218-228, 1938.
8. Jackson, C., and Jackson, C. L.: Tracheotomy, Am. J. Surg. **46**: 519-531, 1939.
9. Lubin, M. L.: Internal Drainage; Its Significance in the Prevention and Treatment of Postoperative Pulmonary Atelectasis, Am. J. Surg. **19**: 80-91, 1933.
10. Mousel, L. H.: Postoperative Atelectasis; the Anesthetist's Part in the Diagnosis and Treatment, J. A. M. A. **115**: 899-902, 1940.
11. Myerson, M. C.: Pulmonary Aspects of Tonsillectomy Under General Anesthesia, Laryngoscope **32**: 929-942, 1922.

EXPERIMENTAL PULMONARY EDEMA FOLLOWING LOBECTOMY AND BLOOD TRANSFUSION

JOHN H. GIBBON, JR., M.D., MARY H. GIBBON (BY INVITATION), AND CHARLES W. KRAUL, A.B. (BY INVITATION)
PHILADELPHIA, PA.

IN 1877 Cohnheim and Lichtheim[2] noted the appearance of acute pulmonary edema in some animals following the administration of massive doses of salt solution intravenously. Warthen[23] more recently studied the effects of administering large amounts of glucose or salt solution intravenously. He stated, "In order to produce pulmonary edema in a dog it was necessary to inject the equivalent of 25 liters of fluid for a human being in thirty-five minutes." It is reasonable to suppose that smaller amounts of intravenous fluid might produce pulmonary edema if the pulmonary vascular bed had been reduced by removal of one or more lobes of the lung. Recently two patients were observed who underwent bilateral lobectomy and died within twelve hours of operation. The following are brief outlines of their clinical courses.

J. M., aged 24 years, had had symptoms of bronchiectasis for eight years. Both lower lobes and the right middle lobe were diseased. On June 5, 1939, the left lower lobe was removed. On Oct. 23, 1940, the right lower and middle lobes were resected under intratracheal ether-oxygen anesthesia. There was some brisk bleeding from a vein in the pulmonary ligament for a short period during the operation. When this occurred, the blood pressure dropped from 130/70 to 80/40. It returned to 110/70 twenty minutes later. The operation required five hours during which the patient received 1,000 c.c. of citrated blood, 800 c.c. of plasma, and 1,000 c.c. of 5 per cent glucose in normal saline. The hemoglobin concentration before operation was 15 Gm. per cent; after operation it was 9.5 Gm. per cent. Postoperatively the patient never regained consciousness. Two hours after operation the blood pressure was normal; the pulse rate was 140 to 160; and the patient had Cheyne-Stokes respiration. He later developed muscular twitching followed by generalized convulsions. The temperature gradually rose to 106° F., and death occurred twelve hours after operation. Shortly after operation he received 1 liter of a one-sixth molar sodium lactate solution because of a mild chill which was attributed to a blood transfusion reaction. He was also given an additional 800 c.c. of plasma between the seventh and twelfth postoperative hours because of the rapid pulse rate and a tendency for the blood pressure to fall. He thus received a total of 4,600 c.c. of fluid intravenously, an amount not excessive for a long abdominal operation associated with some hemorrhage.

L. G.,* aged 19 years, also had had symptoms of bronchiectasis for eight years. Both lower lobes and the right middle lobe were diseased. The left lower lobe was removed Sept. 27, 1939. On Oct. 25, 1940, the right lower lobe was resected under a basal avertin narcosis supplemented by intratracheal nitrous oxide-ether-

From the Harrison Department of Research Surgery, University of Pennsylvania Schools of Medicine, Philadelphia.

Read before the Twenty-Fourth Annual Meeting of the American Association for Thoracic Surgery, Toronto, Canada, June 9, 10, 11, 1941.

*This case is reported through the courtesy of Dr. John B. Flick.

oxygen anesthesia. The right middle lobe, although extensively diseased, was not removed because of the increased operative risk. The operation required four hours during which the patient received 500 c.c. of blood, 1,000 c.c. of plasma and 1,500 c.c. of 5 per cent glucose in normal saline. Postoperatively the patient never regained consciousness. There were gasping respirations and symptoms of air hunger. The pulse rate was 160 per minute, and the blood pressure was normal. The patient died rather suddenly eight and one-half hours after the operation. This patient received a total of 3,000 c.c. of intravenous fluid, again an amount that would not be considered excessive for a prolonged abdominal operation.

Autopsies were performed on both patients. The only significant finding consisted of edema of the remaining lobes. One of the questions which naturally arose was whether the fluids which were administered during and after operation were in any way responsible for the development of the pulmonary edema. The present investigation was undertaken to determine whether intravenous fluids, in amounts well tolerated by normal animals, would produce pulmonary edema when a large part of the functioning pulmonary tissue was removed. This report is concerned only with the effects of blood transfusion following lobectomy. The results following the administration of plasma and salt solution under similar circumstances are being investigated and will be reported later.

METHOD

All the experiments reported here were performed upon cats, anesthetized by an intraperitoneal injection of a 5 per cent solution of sodium amytal (50 mg. of the drug per kilogram of body weight). At the start of all the experiments a large L-shaped glass cannula was tied into the opened trachea in the neck to ensure a free air passage and, in some experiments, to permit the intermittent insufflation of air when the chest was opened. The right femoral vessels were exposed, and blood samples were withdrawn from the right femoral artery through a fine needle into an oiled syringe. The blood was immediately transferred to an oxalated tube under oil and surrounded by ice until it was analyzed for oxygen content and capacity. These determinations were made within one or two hours after the withdrawal of blood. One cubic centimeter of blood from each sample was placed in a Wintrobe hematocrit tube[26] and was centrifuged for a half-hour to determine the volume of the packed red cells. To avoid depletion of the animal's blood volume after withdrawal of the arterial blood sample, the same amount of blood obtained from another cat was injected into the femoral vein. The oxygen analyses were done by the Van Slyke manometric method. All determinations were made in duplicate. The greatest difference between duplicate samples was 0.17 volumes per cent in oxygen capacity and 0.20 volumes per cent in oxygen content.

Blood used for the transfusions and for replacing the blood withdrawn for samples was taken from another cat either the day of the experiment, or twenty-four to forty-eight hours before, with two exceptions. In Experiments 13 and 20 the blood was five days old. When stored, the blood was kept at 5° C. When transfusions were given, the blood was placed in a burette connected by rubber tubing with a glass cannula tied in the femoral vein. The amount given was always 15 c.c. per kilogram of body weight. This was administered over a thirty-minute period with the exception that in some normal control experiments it was given in twenty minutes. References hereafter to "blood transfusion" always refer to this amount of blood given during this time interval. Hematocrit readings were made on samples of the blood transfused. When the arterial blood pressure was recorded, it was read directly through a cannula inserted in the left femoral artery connected to a mercury manometer by rubber tubing filled with a solution of 5 per cent sodium citrate.

In the experiments in which lobectomies were performed a No. 12 French rubber catheter with an open end was threaded into the glass tracheal cannula until its tip lay within the trachea. While the pleural cavities were open, artificial respiration was administered by the intermittent insufflation of air through this catheter. Lobectomies were performed through an incision in the sixth intercostal interspace on either side. The hilar structures of the lobes to be removed were tied with a single ligature of heavy silk. The lobes removed were kept in closed containers to prevent the evaporation of water. A small rubber tube with several perforations near the end was introduced into each pleural cavity through a stab wound in the eighth intercostal space. The incision in the sixth interspace was then closed with a continuous suture, approximating the sixth and seventh ribs. At the conclusion of the operation the catheters draining the pleural cavities were connected to a constant source of 7 cm. of water suction. A T-tube inserted into the suction line was connected with a glass U-shaped water manometer so that respiratory variations in intrapleural pressure could be read. After the chest was closed and suction was established, artificial respiration was stopped and the small rubber catheter was withdrawn from the trachea.

At the conclusion of all experiments an autopsy was performed. Surviving animals were sacrificed by bleeding from the femoral artery. The trachea was clamped, and the thoracic cavity was opened widely by the removal of the sternum and the costal cartilages. Care was taken to avoid bleeding into the pleural cavities until after the presence or absence of fluid had been noted and the amount measured. The color and condition of the lungs or remaining lobes were noted, and the heart and lungs were then removed en masse. The lobes were resected separately by dividing the vessels and bronchi of each lobe close to the pulmonary tissue. The presence or absence of froth in the cut bronchi was noted. The lobes were then weighed to within 0.01 Gm.

RESULTS

The most obvious indication of pulmonary edema is the presence of froth exuding from the orifices of cut bronchi at autopsy. In all of the animals which died from pulmonary edema bubbling of fluid in the trachea could be heard some time before death. Usually pink-tinged froth filled the tracheal cannula and flowed out with expiration. Before these terminal manifestations appeared the arterial blood became noticeably darker. Decreased saturation of the arterial blood with oxygen has been observed to occur with pulmonary edema.[5, 9] The arterial oxygen saturation was determined in all of these experiments. The increased weight of the edematous lobes afforded further confirmation of the diagnosis of pulmonary edema made by inspection at autopsy.

A blood transfusion of 15 c.c. per kilogram of body weight produces no change in the saturation of arterial blood with oxygen in normal cats (Table I). In two experiments, 34 and 35, both pleural cavities were opened and artificial respiration was given for fifty-three and fifty-one minutes, respectively. The pleural cavities were then closed, artificial respiration was stopped, and the blood transfusion was given. In these experiments also the arterial blood was adequately saturated with oxygen after transfusion. Following the transfusion the animals

TABLE I

ARTERIAL BLOOD SAMPLES IN NORMAL CATS WITH TRANSFUSION

EXPERIMENT NO.	SAMPLE A	SAMPLE B		TIME OF SACRIFICE AFTER TRANSFUSION (MIN.)
	OXYGEN SATURATION %	TIME AFTER TRANSFUSION (MIN.)	OXYGEN SATURATION (%)	
15	98	18	97	26
20	99	5	97	29
23	96	12	96	39
29	98	61	97	64
34	97	25	95	120
35	94	10	97	180

were sacrificed at periods varying from one-half hour to three hours. The cats appeared normal in every way at the time of sacrifice, and there was no evidence of cyanosis. At autopsy the lungs appeared grossly normal.

In five animals all the lower lobes and the right middle lobe were removed. This reduces the pulmonary tissue of the cat to approximately 29 per cent of its original mass (Table VI). One animal died one hour and thirty-five minutes after the lobectomy, although the arterial blood showed an almost normal saturation with oxygen twenty-three minutes after the operation (Table II). In the other four animals the arterial blood was adequately saturated with oxygen shortly after lobectomy and as long as four hours later. At the time of sacrifice none of these animals appeared cyanotic. At autopsy the lungs were congested and red, but there was no fluid in the bronchi or evidence of pulmonary edema.

TABLE II

ARTERIAL BLOOD SAMPLES IN EXPERIMENTS WITH LOBECTOMY

EXPERIMENT NO.	SAMPLE A	SAMPLE B		SAMPLE C		TIME OF DEATH (D) OR SACRIFICE (S) AFTER LOBECTOMY (MIN.)
	OXYGEN SATURATION (%)	TIME AFTER LOBECTOMY (MIN.)	OXYGEN SATURATION (%)	TIME AFTER LOBECTOMY (MIN.)	OXYGEN SATURATION (%)	
8	99	23	90			D 99
10	94	5	96	80	97	S 132
11	96	35	99	120	90	S 192
25	96	27	96	248	97	S 280
51	95	31	94	241	93	S 254
52		20	97	255	94	S 280

In nine other animals the same amount of pulmonary tissue was removed and twenty-two to seventy-nine minutes later a blood transfusion was given. Three arterial blood samples were taken during the course of these experiments (Table III). Sample A was taken before the lobectomies; sample B, after the completion of the lobectomies; and sample C, following the transfusion. Samples A and B were all ade-

quately saturated with oxygen. In Experiments 46 and 49 oxygen capacity figures were obtained from sample B, but due to accidental loss there was insufficient blood to determine the oxygen content. Sample C, taken after the transfusion, showed varying degrees of unsaturation with oxygen, with the exception of Experiments 41 and 46. Also, in Experiment 49, in which a blood sample was not obtained after transfusion, the arterial blood appeared bright red at the time of sacrifice, two hours and five minutes after the transfusion. In the other six experiments, during or shortly after the completion of the transfusion,

TABLE III

ARTERIAL BLOOD SAMPLES IN EXPERIMENTS WITH LOBECTOMY AND TRANSFUSION

EXPERIMENT NO.	SAMPLE A	SAMPLE B		SAMPLE C			TIME OF DEATH (D) OR SACRIFICE (S) AFTER SAMPLE C (MIN.)
	OXYGEN SATURATION (%)	TIME AFTER LOBECTOMY (MIN.)	OXYGEN SATURATION (%)	TIME AFTER LOBECTOMY (MIN.)	TIME AFTER TRANSFUSION (MIN.)	OXYGEN SATURATION (%)	
4	97	11	92	110	7	42	D 12
13	95	19	93	117	8	46	D 5
40	94	11	94	89	33	48	D 17
41	95	41	96	180	90	91	S 7
44	97	35	97	139	47	62	D 26
45	96	14	92	152	98	79	S 26
46	95			232	180	93	S 5
49	96						S 125*
50	98	13	93	97	35	71	S 30

*Time after transfusion.

respirations became labored and tracheal râles could be heard. In four experiments the animals died. In three of these four (4, 13, and 40) in which sample C was taken very shortly before death the arterial oxygen saturation was below 50 volumes per cent. In Experiment 44 sample C was taken twenty-six minutes before death and showed a marked, but not as severe a degree of unsaturation. In Experiments 45 and 50 the oxygen saturation of sample C was 79 and 71 volumes per cent, respectively, but at the time of sacrifice twenty-six and thirty minutes later, the arterial blood was of a very dark venous color. In Experiment 41, one of the three in which pulmonary edema did not occur, 38 c.c. of blood were found in the pleural cavities at autopsy. This blood had a hematocrit reading of 36 per cent cells. The cat had been given a transfusion of 43.5 c.c. of blood, with a hematocrit reading of 34 per cent cells. Thus the effect of the transfusion was almost completely lost by hemorrhage into the pleural cavity. In all of the other experiments the amount of fluid found at autopsy in the pleural cavities varied from 4 to 15 c.c., and the hematocrit reading was between 2 and 14 per cent cells.

The lungs of the six animals which had decreased saturation of the arterial blood with oxygen were extremely congested and edematous,

and blood-tinged froth exuded from the cut bronchi. In Experiments 41, 46, and 49, the findings were those of congestion without edema, as in the series of control experiments of lobectomy without transfusion. A possible explanation for the failure of pulmonary edema to develop in Experiments 46 and 49 was found in considering the figures for the oxygen capacity of the blood before and after lobectomy.

In eleven of the fourteen experiments in which the oxygen capacity was determined before and after lobectomy, the capacity was increased (Table IV). The increase was between 0.3 and 6.1 volumes per cent, averaging 2.4 volumes per cent. In the other three experiments there was a decrease of 0.7, 1.1, and 1.4 volumes per cent. In two of these three, 46 and 49, the transfusion failed to produce pulmonary edema or anoxia. In the third, 45, one hour and thirty-nine minutes after transfusion there was only moderate unsaturation, 79 volumes per cent, although, when the animal was sacrificed twenty-six minutes later, the arterial blood was of a dark venous color. Thus in two of the three instances in which the oxygen capacity decreased following lobectomy, blood transfusion did not produce pulmonary edema, while in the third the onset of edema was delayed. Hence it appears that pulmonary edema is more likely to occur if hemoconcentration is present following the lobectomy.

There was generally an increase in the oxygen capacity of the blood following transfusion (Table IV). In Experiment 20 in a normal cat there was a decrease of 0.1 volume per cent, and in Experiment 41, where blood was lost into the pleural cavities, there was a decrease of 0.6 volume per cent. Where the capacity was increased following transfusion after lobectomy, the increase varied between 1.5 and 6.5 volumes per cent, averaging 4.4 volumes per cent. Where the capacity increased in normal cats following transfusion, it varied between 0.8 and 4.6 volumes per cent, averaging 3.1 volumes per cent.

The gross appearance of the lungs of normal cats which received a blood transfusion was similar to that of cats which did not receive a transfusion. In Table V the weights of the lungs are expressed in terms of per cent of body weight. There is no significant increase in the average weight of the lungs of normal cats following transfusion, as compared to the average weight of the lungs of normal cats which were not transfused. The variations also cover the same general range.

In Table VI the weights of the right upper and left upper and middle lobes are compared in three groups of cats; normal cats, cats in which the other lobes were removed, and, finally, cats in which the other lobes were removed and in which a blood transfusion was given. The weights have been expressed in per cent of the weight of the entire lung tissue, and in per cent of the body weight. As would be expected, the most marked differences in the three groups are found when the weights are expressed in terms of body weight. When the weights are

TABLE IV

OXYGEN CAPACITY OF BLOOD

LOBECTOMY				LOBECTOMY AND TRANSFUSION				TRANSFUSION IN NORMAL CATS		
EXPERIMENT NO.	BEFORE LOBECTOMY (VOL. %)	5 TO 35 MINUTES AFTER LOBECTOMY (VOL. %)	80 TO 255 MINUTES AFTER LOBECTOMY (VOL. %)	EXPERIMENT NO.	BEFORE LOBECTOMY (VOL. %)	11 TO 41 MINUTES AFTER LOBECTOMY (VOL. %)	7 TO 180 MINUTES AFTER TRANSFUSION (VOL. %)	EXPERIMENT NO.	BEFORE TRANSFUSION (VOL. %)	AFTER TRANSFUSION (VOL. %)
8	16.1	16.9		4	13.2	19.3	21.0	15	13.9	14.7
10	12.5	15.9	16.3	13	12.4	12.7	16.9	20	17.8	17.7
11	16.4	19.3	20.0	40	10.4	14.7	20.3	23	15.1	19.0
25	21.0	23.0	23.8	41	15.6	18.0	17.4	29	16.7	19.6
51	16.5	18.6	21.1	44	16.0	16.9	23.4	34	13.4	18.0
52		19.3	22.3	45	15.8	14.4	15.9	35	10.0	13.1
				46	11.9	10.8	17.1			
				49	16.4	15.7				
				50	17.5	18.7	24.0			

TABLE V

WEIGHT OF LUNGS EXPRESSED AS PER CENT OF WEIGHT OF BODY WITH AND WITHOUT TRANSFUSION

	VARIATION	AVERAGE
19 Normal cats	0.42 to 0.73	0.53
11 Normal cats following transfusion	0.37 to 0.77	0.58

TABLE VI

WEIGHT OF RIGHT UPPER AND LEFT UPPER AND MIDDLE LOBES EXPRESSED AS PER CENT OF WEIGHT OF LUNGS AND OF BODY

TYPE OF EXPERIMENT	PER CENT OF WEIGHT OF LUNGS		PER CENT OF WEIGHT OF BODY	
	VARIATION	AVERAGE	VARIATION	AVERAGE
19 Normal cats	25 to 34	29	0.12 to 0.22	0.16
Removal of right middle and all lower lobes in 6 cats	37 to 49	42	0.20 to 0.35	0.25
Removal of right middle and all lower lobes and transfusion in 9 cats	46 to 68	53	0.24 to 0.82	0.45

expressed in terms of total lung weight, the edematous lobes increase the total lung weight and thus give a falsely low percentage value for the weights of the upper lobes and the left middle lobe. In terms of body weight removal of the right middle and all the lower lobes results in approximately a 60 per cent increase in the weight of the remaining lobes, whereas, when these same lobes are removed and a transfusion is given, the average weight of the remaining lobes is almost three times the weight of these lobes in normal cats. The increase in weight in the group with lobectomy without transfusion is probably the result of congestion because these lobes were not edematous. In the group with lobectomy and transfusion the increase in the weight represents both congestion and edema.

PRESSURE MEASUREMENTS

In the study of the pathogenesis of the pulmonary edema observed in these experiments, a number of observations were made of the pulmonary arterial, superior vena caval, and systemic arterial pressures. Also in two experiments the left auricular pressure was measured. In all of these experiments heparin (300 units per kilogram of body weight) was given before cannulating the vessels. To measure the pressures in the pulmonary artery and left auricle the heart was exposed in naturally breathing animals by Drinker's method[4] in three instances. In eight other experiments the animals were prepared six weeks in advance by resecting short portions of the two ribs on the left side of the chest overlying the heart, and suturing the pericardium to the opening in the chest wall. At the time of the experiment the heart was exposed by

merely incising the skin and the underlying pericardium.[7] With both these methods the heart is exposed in a naturally breathing animal without opening the pleural cavities. The exposure of the heart to atmospheric pressure results in some diminution of cardiac output.[1] It also increases the superior vena caval and right auricular pressures by an amount approximating the difference between the mean intrapleural pressures in normal animals and the atmospheric pressure. The same increase in pressure also occurs in the pulmonary veins and the left auricle. The elevated pressure in the pulmonary veins would undoubtedly further increase any pulmonary edema that was present or conceivably might initiate pulmonary edema if other factors favoring its development were already present.

A total of eleven experiments was performed in which the pulmonary arterial pressure was measured by means of a special cannula.[21] In three of these the pulmonary arterial pressure was measured in normal cats before, during, and after the standard blood transfusion. Two of these experiments were with Drinker heart preparations and one with a marsupialized pericardium as described above. Prior to the transfusion the pressure ranged from 13 to 20 cm. of water. There was a slight rise during the transfusion, but several minutes later the pressures had returned to their initial level, or slightly above these figures (Table VII and Fig. 1). These pressures are all within the range of those observed previously in cats with Drinker heart preparations.[6]

In eight animals all the lower lobes and the right middle lobe were removed as previously described. The heart was then exposed in one instance (Experiment 18) by Drinker's method and in the other seven by dividing the skin and pericardium overlying the heart in previously prepared animals as described above. The pulmonary arterial pressures in these eight experiments were all above the normal range. In four instances (Experiments 30, 31, 36, and 47) the pressures were very high (Table VII), approximately double the range observed under normal conditions. These four animals died between thirty and ninety minutes after the lobectomies and before a blood transfusion could be given. In the other four experiments (18, 28, 39, and 48) the pressures were not as high, with one exception (Experiment 39). During the course of the transfusion the pressures rose to high levels and did not begin to fall until the animals were moribund (Table VII). In Experiment 48 (Fig. 2) the animal survived the lobectomies for more than two hours before the blood transfusion was begun. The pulmonary arterial pressure which at first was relatively low rose steadily to very high levels during the transfusion, and the animal died one minute after the transfusion was completed.

The failure of these animals to survive the lobectomies without transfusion is probably related to the exposure of the heart to atmospheric pressure. The four animals with lobectomies which were not transfused

(Experiments 30, 31, 36, and 47) all had extensive edema of the remaining lobes at autopsy. As mentioned above, the rise in the pulmonary arterial and the left auricular pressures resulting from exposure of the heart to atmospheric pressure may be responsible for the pulmonary edema.

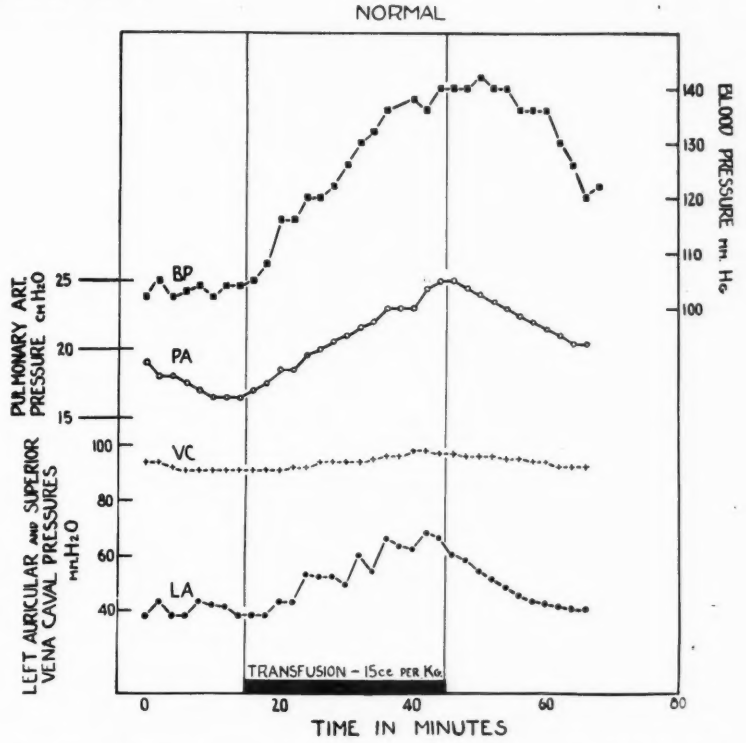

Fig. 1.—Graph of a normal cat receiving a blood transfusion of 15 c.c. per kilogram of body weight over a period of thirty minutes. *BP* is the femoral arterial blood pressure; *PA*, the pulmonary arterial pressure; *VC*, the superior vena caval pressure; and *LA*, the left auricular pressure.

In two normal and three lobectomized cats the right external jugular vein was exposed and a curved stainless steel cannula was passed down into the superior vena cava. The cannula was connected to a saline manometer. In the two normal animals the venous pressure rose slightly during the course of the transfusion and then returned to its previous level (Fig. 1). In the three lobectomized cats the venous pressure rose slowly during the course of the transfusion and then showed an abrupt terminal rise with the failure of the heart just before death (Fig. 2). The left auricular pressure was measured in two experiments. In one normal cat the pressure rose approximately 3 cm. during the course of the transfusion and then returned to its previous level (Fig. 1). In

TABLE VII

PULMONARY ARTERY PRESSURES

NORMAL AND TRANSFUSION				LOBECTOMY			LOBECTOMY AND TRANSFUSION				
EXPERIMENT NO.	PULMONARY ARTERY PRESSURE			EXPERIMENT NO.	INITIAL PULMONARY ARTERY PRESSURE ($CM. H_2O$)	TIME OF DEATH AFTER LOBECTOMY (MIN.)	EXPERIMENT NO.	PULMONARY ARTERY PRESSURE		TIME OF TRANSFUSION AFTER LOBECTOMY (MIN.)	TIME OF DEATH AFTER LOBECTOMY (MIN.)
	INITIAL ($CM. H_2O$)	MAXIMUM ($CM. H_2O$)	FINAL ($CM. H_2O$)					INITIAL ($CM. H_2O$)	MAXIMUM ($CM. H_2O$)		
21	13	17	17	30	56	65	18	33	57	52	91
22	20	24	18	31	44	30	28	32	42	41	67
43	17	25	21	36	54	53	39	41	52	65	98
				47	42	90	48	29	52	132	163

one lobectomized animal there was relatively little change in the left auricular pressure during the transfusion and only a slight rise just before death (Fig. 2). The failure of the left auricular pressure to rise, associated with the marked increase in the pulmonary arterial pressure, appears to indicate some obstruction to blood flow in the pulmonary vascular bed. Lambert and Gremels[11] noted a rise in pulmonary

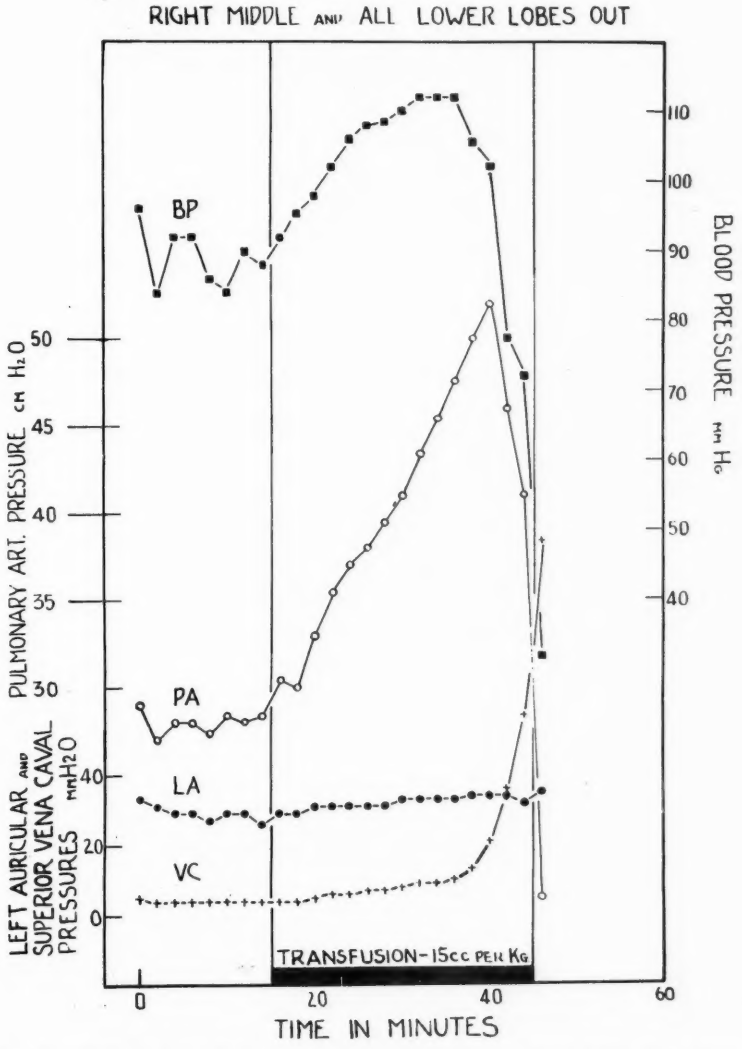

Fig. 2.—Graph of a cat in which the right middle and all lower lobes of the lung had been removed. For symbols see Fig. 1.

artery pressure in the later stages of spontaneously occurring pulmonary edema in heart-lung preparations and attributed it to the mechanical influence of the fluid in the alveoli upon pulmonary capillaries. The changes in systemic arterial blood pressure with transfusion in normal and lobectomized cats were always similar to those illustrated in Figs. 1 and 2.

DISCUSSION

The factors concerned in the pathogenesis of pulmonary edema have recently been reviewed by Luisada.[17] There are, however, certain points which require emphasis. It is obvious that the conditions which favor edema in other parts of the body will also tend to favor the accumulation of fluid in the lungs. The hypothesis of Starling[20] that the movement of fluid in and out of the tissue is influenced by the colloid osmotic pressure of the blood and the capillary blood pressure is generally accepted. Injury to the capillary wall, which under normal conditions is relatively impermeable to protein,[13] results in the filtration of all the constituents of the plasma into the tissue spaces and the appearance of edema. Increased capillary pressure, decreased colloid osmotic pressure of the plasma, and injury to the capillary endothelium are the three common causes of edema and must operate in the lung as well as in other parts of the body.

Pulmonary edema arising from increased capillary pressure as the result of left-sided heart failure is a well-known clinical entity which was first recognized by Welch[25] in 1878 and has been adequately discussed and studied in recent years by Harrison and his co-workers[9] and Weiss and Rob.[24] Decreased colloid osmotic pressure alone is probably rarely responsible for the development of edema. The pulmonary edema resulting from the inhalation of poisonous gases such as phosgene is a familiar example of the type due to direct capillary injury.[3]

One of the peculiar characteristics of pulmonary edema is its occasional abrupt onset and rapid progression. There are probably many factors which are responsible for the characteristics. A sudden onset is frequently associated with pulmonary edema due to left ventricular failure. It is easy to comprehend that even a very short-lived and minute decrease in the output of the left ventricle, compared to that of the right, would result in severe pulmonary congestion with increased capillary pressure and a tendency to edema. It is, indeed, surprising that the condition is not more frequently seen in view of the delicate balance which must be constantly maintained between the outputs of the left and right sides of the heart.

The rapid progress, on the other hand, is undoubtedly dependent on several other factors. The pulmonary edema itself results in increased respiratory effort. Fraser and his co-workers[5] in 1928 suggested that this increase in respiratory movements might lead to further pulmonary

congestion which would tend to aggravate the edema. Langeron[15] has recently restated this conception. Harrison and his associates[9] reported a patient with pulmonary edema in whom the saturation of the arterial blood with oxygen rose from 56 to 80 per cent following the administration of morphine. It is possible that the improvement resulted from the decreased respiratory activity. Grollman[8] in 1930 showed that voluntary overventilation in normal men resulted in an increase in cardiac output, and Harrison and his co-workers[9] confirmed this observation. An increased cardiac output in the presence of edema would tend to aggravate the edema.

In the experiments reported here the pulmonary edema was always accompanied by a marked rise of the pressure in the pulmonary artery to more than double the normal level. Lambert and Gremels[11] in 1926 also observed this rise in pulmonary arterial pressure in the spontaneously occurring edema of heart-lung preparations. Using a method by which they determined the electrical conductivity of the lungs, they found that the pulmonary edema occurred prior to the rise in the pressure of the pulmonary artery. They attributed the marked rise of the pulmonary arterial pressure in the late stages of edema to the mechanical influence of the fluid in the alveoli on the pulmonary capillaries. The absence of failure of the left side of the heart in the experiments reported here makes it evident that the rise in pulmonary artery pressure is not due to any back-pressure effect through the pulmonary vascular tree.

Fraser and his co-workers[5] noted a decreased saturation of the arterial blood with oxygen in a patient with pulmonary edema from congestive heart failure, and Harrison and his associates[9] confirmed this observation. In the experiments reported here the decreased saturation of the arterial blood with oxygen was regularly observed when severe pulmonary edema was present. As anoxemia increases capillary permeability,[12] a vicious circle is established which tends to increase the edema already present.

Miller[19] has shown that the lymphatic vessels of the lung do not extend beyond the alveolar ducts. Lorber[16] recently reported some evidence that this anatomic characteristic may favor the rapid development of edema. In heart-lung preparations the lymph flow from the lung was measured during the development of spontaneous pulmonary edema. He found that there was a steady decrease in the lymph flow. In the extremities, on the other hand, it is probable that large accumulations of fluid in the tissue spaces are removed primarily through the lymphatic channels.[10]

In most tissues and organs of the body the tissue pressure resists the rapid accumulation of fluid in the extracellular spaces. Direct measurement of tissue pressure is difficult. Meyer and Holland[18] found a pressure of 7 cm. of water in the skin and 3 cm. in the subcutaneous tissues.

Krogh, Landis, and Turner[10] noted that venous pressures below 15 cm. of water failed to produce measurable filtration of fluid into the tissues. Similarly, Landis and Gibbon[14] found no significant filtration of fluid into the forearm with venous pressures below 10 cm. of water. Thompson and his co-workers[22] noticed that fluid was lost from the blood during quiet standing, but the loss ceased before edema appeared in the legs. Landis and Gibbon[14] showed that the accumulation of fluid in the forearm decreased the rate of filtration into the tissue at any given venous pressure. Thus as fluid accumulates in the tissues, the increase in tissue pressure retards the further filtration of fluid from the capillaries. Until the alveoli become entirely filled with fluid no such inhibiting factor operates in the lungs. The delicate alveolar walls surrounding small air spaces offer little or no resistance to the outpouring of fluid from the pulmonary capillaries. Only air needs to be displaced to accommodate the fluid. Thus the failure to develop an increased tissue pressure during the filtration of excessive amounts of fluid from the pulmonary capillaries may contribute significantly to pulmonary edema.

SUMMARY

1. A blood transfusion of 15 c.c. per kilogram of body weight was given to six normal cats at a constant rate over a period of one-half hour. The saturation of the arterial blood with oxygen was normal before and after the transfusion, and at autopsy there was no evidence of pulmonary edema.

2. The right middle and all lower lobes of the lungs were removed from six cats. The saturation of the arterial blood with oxygen was normal before the lobectomies and as long as four hours after the operation. At autopsy the remaining lobes were found to be congested but not edematous.

3. In nine animals after the removal of the same lobes of the lung, a blood transfusion of 15 c.c. per kilogram of body weight was given. Most of the animals showed varying degrees of unsaturation of the arterial blood with oxygen, and four of them died shortly after the completion of the transfusion.

4. The oxygen capacity of the blood was determined in fourteen cats. It was found to be increased in eleven instances after the removal of all the lower lobes and the right middle lobe. In two of the three cats in which increased capacity did not occur after lobectomy, subsequent blood transfusion failed to produce evidence of anoxemia, and in the third instance the anoxemia did not appear for some time following transfusion and was of a mild degree.

5. A blood transfusion of 15 c.c. per kilogram of body weight did not produce pulmonary congestion or edema in normal cats as evidenced by the appearance and weight of the lungs at autopsy.

6. Pulmonary edema did not follow lobectomy as described above, although the remaining lobes were congested and weighed half again as much as the same lobes in normal cats.

7. When a blood transfusion was given after completion of the lobectomies, pulmonary edema occurred in the majority of instances. Pink froth was frequently observed filling the trachea shortly before death, and at autopsy similar frothy fluid exuded from the cut bronchi. The weight of these edematous lobes was approximately three times that of the corresponding lobes in normal cats.

8. In normal cats the standard blood transfusion produced a transient increase in the systemic arterial, the pulmonary arterial, the left auricular, and superior vena caval pressures. In the lobectomized cats the pulmonary arterial pressure was found to be always well above the normal range. Blood transfusion resulted in a further increase and death. The systemic arterial and the venous pressures in the lobectomized cats with transfusion were unchanged, or showed a slight rise, until the animal was moribund, when the venous pressure rose and arterial pressure fell.

CONCLUSION

In cats in which there has been an acute reduction in pulmonary tissue, a blood transfusion of a size well tolerated by normal animals may produce pulmonary edema, decreased saturation of the arterial blood with oxygen, and death.

REFERENCES

1. Beck, C. S., and Cox, W. V.: The Effect of Pericardiostomy on the Mechanics of the Circulation, Arch. Surg. **21**: 1023, 1930.
2. Cohnheim, J., and Lichtheim, L.: Ueber Hydrämie und Hydrämisches Oedem, Arch. f. path. Anat. u. Physiol. u. f. klin. Med. **69**: 106, 1877.
3. Cordier, D., and Francais, J.: Modifications de la fonction respiratoire du sang au cours de l'oedème pulmonaire expérimental; étude des variations de la courbe de saturation en oxygène et de la marche de l'oxygénation et de la réduction du sang artériel, Ann. de physiol. **15**: 339, 1939.
4. Drinker, C. K.: A Useful Heart Method, J. Exper. Med. **33**: 675, 1921,
5. Fraser, F. R., Harris, C. F., Hilton, R., and Linder, G. C.: Arterial Carbon Dioxide Pressure in Cardiac Dyspnea, Quart. J. Med. **22**: 1, 1928.
6. Gibbon, J. H., Jr., and Churchill, E. D.: Changes in the Pulmonary Circulation Induced by Experimentally Produced Arteriovenous Fistula, Arch. Surg. **21**: 1188, 1930.
7. Gibbon, J. H., Jr.: The Maintenance of Life During Experimental Occlusion of the Pulmonary Artery Followed by Survival, Surg., Gynec. & Obst. **69**: 602, 1939.
8. Grollman, A.: The Effect of Breathing Carbon Dioxide and of Voluntary Forced Ventilation on the Cardiac Output in Man, Am. J. Physiol. **94**: 287, 1930.
9. Harrison, T. R., Calhoun, J. A., and Harrison, W. G., Jr.: Congestive Heart Failure; Observations Concerning Mechanism of Cardiac Asthma, Arch. Int. Med. **53**: 911, 1934.
10. Krogh, A., Landis, E. M., and Turner, A. H.: The Movement of Fluid Through the Human Capillary Wall in Relation to Venous Pressure and to the Colloid Osmotic Pressure of the Blood, J. Clin. Investigation **11**: 63, 1932.
11. Lambert, R. K., and Gremels, H.: On Factors Concerned in the Production of Pulmonary Edema, J. Physiol. **61**: 98, 1926.

12. Landis, E. M.: Micro-Injection Studies on Capillary Permeability. III. The Effect of Lack of Oxygen on the Permeability of the Capillary Wall to Fluid and to the Plasma Proteins, Am. J. Physiol. **83:** 528, 1928.
13. Landis, E. M.: Capillary Pressure and Capillary Permeability, Physiol. Rev. **14:** 404, 1934.
14. Landis, E. M., and Gibbon, J. H., Jr.: The Effects of Temperature and of Tissue Pressure on the Movement of Fluid Through the Human Capillary Wall, J. Clin. Investigation **12:** 105, 1933.
15. Langeron, L.: Sur la pathogénie des oedèmes aigus du poumon, Gaz. d. hôp. **109:** 601, 1936.
16. Lorber, V.: Lymph Flow From Heart-Lung Preparation During Pulmonary Edema, Proc. Soc. Exper. Biol. & Med. **43:** 170, 1940.
17. Luisada, A.: The Pathogenesis of Paroxysmal Pulmonary Edema, Medicine **19:** 475, 1940.
18. Meyer, F., and Holland, G.: Die Messung des Druckes in Geweben, Arch. f. exper. Path. u. Pharmakol. **168:** 580, 1932.
19. Miller, W. S.: The Lung, Springfield, Ill., 1936, Charles C Thomas.
20. Starling, E. H.: On the Absorption of Fluids From the Connective Tissue Spaces, J. Physiol. **19:** 312, 1895-96.
21. Swift, W. E., Haggart, G. E., and Drinker, C. K.: A New Method for Measuring the Pressure in the Pulmonary Artery, J. Exper. Med. **36:** 329, 1922.
22. Thompson, W. O., Thompson, P. K., and Dailey, M. E.: The Effect of Posture Upon the Composition and Volume of the Blood in Man, J. Clin. Investigation **5:** 573, 1927.
23. Warthen, H. J.: Massive Intravenous Injections, Arch. Surg. **30:** 199, 1935.
24. Weiss, S., and Robb, G. P.: Cardiac Asthma (Paroxysmal Cardiac Dyspnea) and Syndrome of Left Ventricular Failure, J. A. M. A. **100:** 1841, 1933.
25. Welch, W. H.: Sur Pathologie des Lungenödems, Virchows Arch. f. path. Anat. **72:** 375, 1878.
26. Wintrobe, M. J.: A Simple and Accurate Hematocrit, J. Lab. & Clin. Med. **15:** 287, 1929.

DISCUSSION

DR. EVARTS A. GRAHAM, St. Louis, Mo.—I have nothing to say, except that I think it would be a shame to have this very beautiful and important demonstration passed by without some comment. I feel certain that Dr. Gibbon has put his finger on the cause of many of the tragedies which occur after total pneumonectomy, and to some extent after lobectomy when we overexert ourselves, perhaps, to try to prevent the patient from suffering from shock by overloading the blood stream.

I was not quite sure about one point. I did not hear how much blood was used for transfusion of these cats from which one or two lobes had been removed. That is to say, what proportion of body weight was used? How did that compare with human blood transfusion, for example?

DR. LEO ELOESSER, San Francisco, Calif.—I have one question. I wonder whether Dr. Gibbon could tell us whether death was occasioned by the tying off of the pulmonary vessels, or by the removal of lung as such and whether it would be worthwhile to tie the pulmonary vessels singly and see if the same thing might happen.

DR. H. BRODIE STEPHENS, San Francisco, Calif.—My comments are not meant to criticize this presentation; but for my own interest, I should like to know why Dr. Gibbon removed so much of the functioning lung. Have experiments been carried out in which one lobe, then two or three lobes were removed and the effect of blood transfusion in these animals observed?

It occurred to me that the experimental work presented was not exactly comparable to our clinical practice, in that more lung was removed in the experimental animal than is the common procedure when dealing with the human subject.

DR. EDWARD D. CHURCHILL, Boston, Mass.—I should like to ask Dr. Gibbon whether the increase in the oxygen capacity indicated hemoconcentration, and if that constitutes an indication for plasma transfusion rather than whole blood transfusion.

DR. HARRY R. DECKER, Pittsburgh, Pa.—I should like to ask Dr. Gibbon whether there was any evidence at autopsy in the two cases of renal damage and, if so, whether perhaps this effect may not have been a contributory factor in the fatalities.

DR. M. DAWSON TYSON, Hanover, N. H.—I should like to hear what Dr. Gibbon has to say about the x-ray appearance in human beings after administration of large amounts of fluid following pulmonary operation.

We have been in the habit of taking rather frequent x-rays if we felt the patient needed large amounts of fluid, and we feel we are able to pick up pulmonary congestion before the severe edema has developed.

DR. JOHN H. GIBBON, JR.—In reply to Dr. Graham's question, I did state, perhaps not clearly enough, that the transfusion is 15 c.c. of blood per kilogram of body weight, given over a period of a half hour. That corresponds to a transfusion in man of about 1,000 c.c.

Dr. Eloesser asked whether it would make any difference if we tied just the vessel and not the bronchi. I believe it would undoubtedly have the same effect; that is, all the output of the right heart would still have to pass through a much smaller vascular bed.

Dr. Stephens asked why we took out so much pulmonary tissue. We have been interested in the cases in which both lower lobes have been removed in human beings and found only a few reports, Dr. Eloesser's, Dr. Overholt's, Dr. Churchill's, Dr. Graham's, and a few others, but we also felt there were probably a great many patients who died following bilateral lower lobectomy and had not been reported.

We wanted to produce a rather extensive diminution in pulmonary tissue, and we thought that we could best do it by removing both lower lobes and the right middle lobe. In normal cats, you can produce pulmonary edema if you administer large volumes of intravenous fluid. I do not doubt that, if we took out only one lower lobe, we would have to employ larger amounts of intravenous fluid to produce pulmonary edema than proved necessary when both lower and the right middle lobes had been removed.

Dr. Churchill asked about the oxygen capacity. We believe it does represent hemoconcentration. The volume of the packed red cells also increased following lobectomy. Why that occurs, I am not sure. In the few instances in which it did not occur, in which perhaps a certain amount of bleeding took place during the operation, the transfusion did not produce edema.

We are investigating the effects of plasma and intravenous salt solution but are not prepared to report on them. When hemoconcentration is present, plasma is undoubtedly a more suitable fluid to use than blood.

THE "SNIFF TEST" IN THORACIC SURGERY

With a Review of 119 Phrenic Nerve Interruptions*

Jack Greenfield, M.D. (by invitation), Cleveland, Ohio, and
George M. Curtis, M.D., Columbus, Ohio

THE "sniff test" reveals the presence of paralysis of the hemidiaphragm following phrenic nerve interruption from any cause. It is of particular significance since interruption of the phrenic nerve has become established as a therapeutic measure as well as an adjunct to more radical thoracic surgery.

Phrenic nerve interruption as a method of treatment has become deservedly popular in the management of certain patients with pulmonary tuberculosis.[1, 5, 6, 22] It was Stuertz, in 1911, who first suggested paralyzing one-half of the diaphragm by division of the phrenic nerve in the base of the neck, in order to relax the diseased lower lobe which could not be compressed by artificial pneumothorax.[30] By 1913, Sauerbruch had reported five patients for whom he had performed phrenicotomy.[13] Soon the disadvantages of simple section of the nerve began to appear, and Walther, in 1914, showed that simple phrenicotomy did not completely paralyze the diaphragm in more than 50 per cent of the cases and that in 20 per cent normal function returned.[14] Phrenic nerve interruption, however, was not to lapse as a therapeutic measure.

Goetze, in 1922, showed that in from 20 to 80 per cent of the instances examined there was more than one phrenic nerve. In fact, he thought that the sympathetic plexus also sent motor fibers to the diaphragm along the phrenic nerve. Consequently, he proposed phrenicectomy with section of the connecting sympathetic fibers and exposure of the nerve to the subclavius muscle, since this nearly always contains the accessory phrenic nerves when they are present.[15] Since, even then, uniform success in obtaining paralysis of the hemidiaphragm was not accomplished, Felix, in 1922, proposed phrenic exeresis or evulsion of the nerve to ensure the removal of all motor fibers.[31] Phrenic nerve interruption by phrenicectomy or exeresis was then rapidly taken up on the Continent as a valuable therapeutic measure. Nevertheless, it was not until 1925, when Alexander introduced it, that it became popular in the United States.[16]

It was later shown by Kiss and Ballon that the sympathetic fibers carry only vasomotor impulses to the diaphragm and perhaps that they have some trophic function, but no motor effect. The same authors showed that the phrenic nerve was both motor and sensory and that

From the Department of Research Surgery, The Ohio State University.

Read (by title) before the Twenty-Fourth Annual Meeting of the American Association for Thoracic Surgery at Toronto, Canada, June 9, 10, and 11, 1941.

*This investigation was aided by a grant from the Comly Fund for Research of The Ohio State University.

some of the sensory fibers were supplied by the eleventh and twelfth thoracic nerves at the costal attachments of the diaphragm.[21]

Because exeresis presents, though infrequently, an element of danger,[10] and since only temporary paralysis of the hemidiaphragm is frequently desired, phrenemphraxis or phrenic crush has more recently become the more popular method.[1, 15] Given a series of patients with proper indications, the accomplishment of a successful paralysis of the hemidiaphragm should yield satisfactory results in at least two-thirds and even on up to 80 per cent.

The best method which we have found for demonstrating a successful paralysis of the hemidiaphragm is the "sniff test." This is also of proved value in following the gradual return of function of the diaphragm in those patients for whom temporary interruption of the phrenic nerve has been accomplished. The principles underlying the selection of cases, the reasons for the satisfactory results, and the basis for the test which will determine a successful paralysis may be understood best when one is acquainted with the anatomic and physiologic effects of the operation on the diaphragm, the intrapleural space, and the involved lung.

Upon interruption of the phrenic nerve and its principal accessories, the paralyzed hemidiaphragm ascends into the thorax, reducing the chest capacity on the paralyzed side with a resultant compression of the lung. Brunner, in his experiments,[25] showed that the collapse provided by hemidiaphragmatic paralysis can result in a reduction of from one-sixth to one-third of the total lung volume. Matson, in comparing this effect with pneumothorax therapy, found that the resultant compression of the lung was equal to that following the introduction of from 400 to 800 c.c. of air into the pleural cavity.[22] Yet, even in the face of this corresponding decrease in vital capacity, Werner found no decrease in the oxygen consumption.[19]

Another result is that the diaphragm no longer regulates in its normal manner the changes in intrathoracic pressure during the succeeding phases of respiration, but responds in a more passive manner to those pressures in so far as certain pathologic changes in the diaphragm and pleura permit. On inspiration, the negative pressure is less on the paralyzed side. A functioning hemidiaphragm makes approximately 20,000 respiratory excursions in twenty-four hours. This ceases upon paralysis thus relieving the lung of a corresponding amount of movement.

The rise is usually less marked and often may be entirely absent in patients with diaphragmatic adhesions. Cooper[26] reported no rise in 38 out of 103 cases after phrenic exeresis; nevertheless, the downward inspiratory pull of the diaphragm ceased. The gradual rise of the diaphragm may continue to some degree for from six months to a year, as the tone of the paralyzed muscle is gradually lost. The musculature

undergoes an eventual atrophy leaving a thin fibrous structure. Histologically, fatty degeneration is present, an occasional atrophic muscle cell is seen, and the fibrous element is thinner than normal. Its tensile strength, however, remains good, and the dangers of later herniation are slight.[7] This gradual rise of the diaphragm is further accentuated by shrinkage of the pulmonary scar tissue as healing in the lung progresses. The maximum rise may not be attained for a year or more and may be as high as 13 cm.[22]

The effects on the lung are several. There is rest by cessation of the force of diaphragmatic action, and compression due to the decrease in size of the thoracic cage. Forsee, among others, claims that relaxation of the lung is greatest where the tendency to shrinkage is greatest.[5, 22] This tendency appears to be at a maximum where cavities and tubercles are healing and, therefore, paralysis of the diaphragm has even a selective effect with respect to cavity collapse.

An interesting effect of unilateral paralysis of the diaphragm is a resultant paradoxical motion, which has been noticed by many.[5, 8, 12, 22] This is frequently seen upon fluoroscopy after interruption of the phrenic nerve. The paralyzed hemidiaphragm *rises* on deep inspiration while the unparalyzed leaf descends. Paradoxical motion of this type was first recognized and described by Kienböck in 1898.[32] By fluoroscopy he observed that in a unilateral pyopneumothorax the diaphragm with the supernatant fluid *ascended* during inspiration while the other diaphragm descended normally. He made the erroneous assumption that the diaphragm was paralyzed and his explanation of the phenomenon, which he called paradoxical motion, was that the affected hemidiaphragm was forced up during inspiration by the increased abdominal pressure caused by the descent of the unaffected leaf.

Vadone and Udaondo analyzed the Kienböck phenomenon.[27, 28] They found that paradoxical movement of the diaphragm sometimes accompanies pneumothorax, but that in those instances it is always associated with an increased positive pressure and does not occur if the intrapleural pressure at expiration remains negative. When paradoxical motion did occur in the presence of pneumothorax it disappeared when the patient lay on that side. If the phrenic nerve to that side is interrupted, however, paradoxical motion will occur in any position.

Sauerbruch[9] and later Meyer[10] stated that the cause of paradoxical motion of the diaphragm after phrenic nerve interruption is that during inspiration the diaphragm on the paralyzed side is sucked up due to elevation of the thoracic wall; while during expiration, on account of the increase in intrathoracic pressure, it is pushed down. However, the increase in abdominal pressure, as well as the suction by negative intrathoracic pressure on the affected side, appears to play a part in the production of the phenomenon. The effect may even be aided by the

pendelluft action of Brauer[11] which on inspiration, due to the greater negative pressure on the unaffected side, causes air to be drawn out of the partially compressed lung, further collapsing it. This, in turn, increases the negative pressure on the affected side and sucks the diaphragm up even further (Fig. 1). On expiration it forces air into the affected lung, expanding it. Thus, the secondary flutterings frequently seen after the initial rise on inspiration are to be explained.

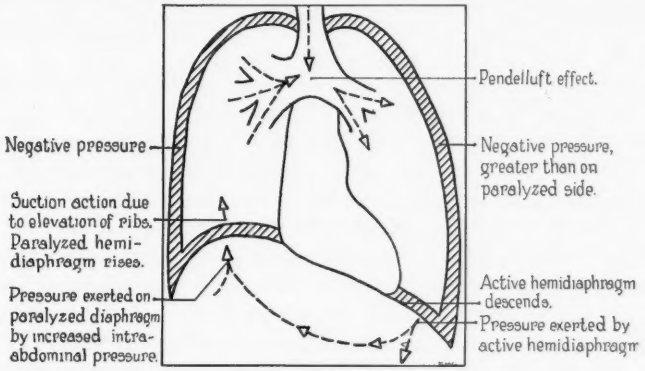
Pendelluft effect.
Negative pressure
Negative pressure, greater than on paralyzed side.
Suction action due to elevation of ribs. Paralyzed hemidiaphragm rises.
Active hemidiaphragm descends.
Pressure exerted on paralyzed diaphragm by increased intra-abdominal pressure.
Pressure exerted by active hemidiaphragm

Fig. 1.—Diagrammatic presentation of the factors involved in the production of a positive "sniff test."

The thoracic surgeon, in his effort to determine whether he has obtained complete motor interruption to the diaphragm, has attempted several methods. The majority observe the resultant elevation of the diaphragm either by fluoroscopy or roentgenogram, or the fixation of the diaphragm, or both. As shown by Cooper, the rise may not occur in from 35 to 38 per cent of the patients, depending upon the condition of the lung, adhesions, fibrosis, etc. Also, the diaphragm may not rise until later. Although fixation following phrenic nerve interruption is fairly constant, instances are recorded in which the normally moving diaphragm by its increased activity has caused motion on the paralyzed side.[8]

In searching for a better indication as to whether interruption of all the motor branches to the hemidiaphragm had been obtained, Walther was the first to study the results by fluoroscopy, using the paradoxical motion resultant to forced respiration as a basis for demonstrating a successful paralysis.[14] As a further improvement, since paradoxical motion did not always occur with forced respiration, Bittdorf introduced a test by which the paradoxical motion could be demonstrated even better.[24] In order to elicit the sign, the patient was instructed to close the nostrils with the fingers and then attempt to take a deep breath with the mouth closed. The paralyzed hemidiaphragm rose in the thoracic cavity; on the noninterrupted side it descended.

A sensitive and more easily applied test is the "sniff test," first described by Hitzenberger in 1927, and elicited by directing the patient suddenly to sniff.[23] The paralyzed hemidiaphragm rises whereas the normal descends. In the presence of hydrothorax, thickened pleura, or extensive disease of the lung, visibility under the fluoroscope may be so obscured that even this test may not be conclusive. In the presence of diaphragmatic hernia, because the positive pressure in the abdomen and the negative pressure in the chest cannot exert their influence in the presence of a large opening in the hemidiaphragm, this test cannot be elicited.

Phrenic nerve interruption may be obtained by any one of several methods. The phrenic nerve may be temporarily interrupted by simple section with immediate suture, from which regeneration may be expected within four months.[2] Regeneration following alcohol injection, as described by Goetze, occurs in about three months.[3] Freezing with ethyl chloride delays the return of function for about eight days. The return of function subsequent to novocain injection ensues in from six to twenty-four hours. Phrenemphraxis, with exposure of the nerve and crushing of a segment together with section of the accessory nerves, delays the return of function for from four to eighteen months. The latter is the most popular method in use today. For permanent effects, phrenicectomy as proposed by Goetze[15] or the more radical and successful exeresis of the nerve as devised by Felix[31] are employed.

The unsatisfactory results sometimes obtained are due to abnormal positions of the phrenic nerve, multiple nerves, and the presence of accessories. This has been ably discussed and illustrated by Matson.[22] Accessory divisions arise most commonly from the fifth cervical nerve, either near or along the nerve to the subclavious muscle. If arising with the nerve to the subclavius, the accessory division usually separates from it just before the nerve enters the muscle. The accessory branch then continues downward, passing in front of the subclavian vein, to join the main stem of the phrenic nerve, usually just beyond the sternal end of the first rib. Accessory branches, however, may arise from the second, third, sixth, seventh, or eighth cervical trunks. These anomalies make it imperative to obtain good exposure of the anterior scalene muscle and to explore each side in addition to the anterior surface. Sometimes, when phrenic crush is attempted, the crushing is inadequate and although a positive test for paralysis is obtained immediately after operation, function returns a few days later.

Keeping the principles and effect of phrenic nerve interruption in mind, the thoracic surgeon can find many uses for the operation not only in treatment per se, but as a valuable adjunct to other more complex surgical procedures.

The indications for phrenic interruption in pulmonary tuberculosis are found largely in patients with unilateral disease who do not respond

well after several months of the usual bed-rest treatment, those who have persistent open cavities with positive sputum, or those who have a constant cough with expectoration. In the majority of cases pneumothorax has been tried without success. Phrenic interruption is likewise employed as an adjunct where, subsequent to pneumothorax, basilar adhesions prevent sufficient collapse and, finally, either preceding or following thoracoplasty.[1, 5, 12]

It is also of value in the collapse of chronic empyema cavities which are being drained,[12] as a palliative measure in diaphragmatic hernia when direct operative intervention is contraindicated, and as a preliminary measure to the radical cure of diaphragmatic hernia.[4, 18] It can be used as an aid in obliterating residual cavities remaining after the removal of tumors of the thoracic wall,[18] after lobectomy,[33] and after curettage or dissection of persistent bronchial fistula.[12] Occasionally phrenic interruption has been employed to relieve intractable hiccough,[17, 29] or to stop acute or repeated pulmonary hemorrhages both in pulmonary tuberculosis and bronchiectasis.[12] It may also be employed in collapsing the residual cavity after drainage of a lung abscess.

It is difficult to measure the results of phrenic interruption quantitatively; however, they may be considered satisfactory when the desired effect is obtained. Thus, if an empyema cavity is made smaller, if an incomplete collapse of a lung is completed, or if the desired decrease in sputum production is obtained, the result may be recorded as satisfactory. Trudeau obtained 66 per cent improvement in 300 cases of pulmonary tuberculosis[1] and Cooper reported contraction of cavities in 37 per cent of 103 cases. When used as an adjunct to multiple-stage thoracoplasty, it may control lower lobe lesions and thereby decrease the number of ribs to be resected.[1, 6] Moreover, if temporary interruption is employed, that much vital capacity is preserved for the future benefit of the patient.

From October, 1933 to July, 1939, 119 phrenic nerve interruptions were performed on the research surgical service, for pulmonary tuberculosis with hemorrhage, because of unsuccessful pneumothorax therapy, as an aid to pneumothorax where basal adhesions prevented adequate collapse, as sole therapy in pulmonary tuberculosis, as an aid to collapse by thoracoplasty, to facilitate the operation for diaphragmatic hernia, for intractable hiccough, to aid in the closure of bronchial fistulas and drained lung abscesses, and to aid in the obliteration of empyema cavities (Table I). For the purposes of a brief report of this experience, measurements of the vital capacity and tests of diaphragmatic function were made and the results judged within one month following the interruption.

Three types of interruption were accomplished, exeresis for thirty-one patients, crush for eighty-seven, and novocain injection in one instance. Fluoroscopic examination was not made of seven patients because they

TABLE I

THE INDICATIONS FOR AND THE RESULTS OF 119 CONSECUTIVE PHRENIC NERVE INTERRUPTIONS

INDICATION FOR PHRENIC NERVE INTERRUPTION	TYPE OF INTERRUPTION			FLUOROSCOPIC EXAMINATION						RESULTS		REMARKS
				POSITION OF DIAPHRAGM				SNIFF TEST				
	EXERESIS	CRUSH	NOVOCAIN INJECTION	IMMOBILE	MOBILE	ELEVATED	NOT ELEVATED	POSITIVE	NEGATIVE	SATISFACTORY	UNSATISFACTORY	
Pulmonary tuberculosis with hemorrhage	1	4		3		3		3		5		2 not tested due to illness
Unsuccessful pneumothorax therapy	3	12		15		10	5	14	1	13	2	
Aid to pneumothorax therapy	6	17		22		18	4	22		21	2	1 not tested due to illness
Aid to thoracoplasty	11	22		27	3	25	5	27	3	22	12	3 not tested due to illness
Sole therapy for pulmonary tuberculosis	10	24		33	1	22	12	33	1	25	8	Diaphragm not visualized due to thick pleura
To facilitate operation for diaphragmatic hernia		1		1		1				1		Desired immobility demonstrated at operation
The control of hiccough		1	1							2		2 not tested due to illness
To aid in the closure of bronchial fistula		2		2		2		2		1	1	
To aid in the closure of drained lung abscess		2		2		2		2		2		
To aid in the closure of empyema cavity after lobectomy		2		2		2		2		2		
Totals	31	87	1	107	4	85	26	105	5	94	25	5 (4%) were unsuccessful

were too ill to take to the fluoroscope; in one patient the diaphragm could not be visualized and in one, the patient with diaphragmatic hernia, neither a negative nor a postive "sniff test" could be obtained.

Of the 111 patients examined, the diaphragm was found to be immobile in 107, mobile in 4, elevated in 85, and not elevated in 26. The "sniff test" was positive in 105 and negative in 5. All the negatives proved to be due to lack of complete motor paralysis. In one case the hemidiaphragm moved in spite of paralysis as proved by the ultimate rise and positive "sniff test." The immediate results were satisfactory in 94, or 79 per cent, of the total number of patients treated, while the "sniff test" was applicable to 110 of the 111 cases tested.

The anatomy encountered at operation was normal in 93 and abnormal in 26 patients; 28 had accessory phrenic nerves; 1 had a double and 1 a triple phrenic nerve. Of the 28 with accessories, 8 had two and 1 had three accessory branches. In the patients with a negative "sniff test," it became apparent that accessories had been missed.

In 25 interruptions, where no other operations were done or any other affecting therapy instituted, the vital capacity was measured before and after operation. The immediate results were as follows: The average decrease in vital capacity was 363 c.c., the greatest single decrease being 785 c.c. The lowest single percentage decrease was 7 per cent and the greatest was 30 per cent of the total vital capacity.

Of all the interruptions, ten were recrushes, one because of failure due to inadequate exploration at the first operation and nine because of return of function before the desired effects were obtained, from six to eighteen months subsequent to the primary interruption.

Phrenic nerve interruption is of proved value in certain intrathoracic lesions. Exeresis may be done if permanent paralysis is desired. Although certain of our patients had immediate reactions characterized by mild shock or gastric symptoms of dyspnea, they soon recovered and there was no morbidity or mortality. A wiser procedure in most cases is phrenemphraxis or phrenic crush, because of its simplicity and since a temporary paralysis is often desired. The effects last from four to eighteen months and, if necessary, recrush may then be accomplished. Adequate exposure and careful exploration for accessory and accompanying phrenic nerves is necessary.

A valuable and readily accomplished test for the resultant diaphragmatic paralysis is the "sniff test." This is of particular service in following the return of function in patients treated by temporary phrenic interruption. The test is based upon the paradoxical motion of the diaphragm which sometimes occurs upon forced respiration following paralysis of the hemidiaphragm, but which always occurs when no dissipation of the inspired air is permitted by closure of the glottis after the forced inspiration during a sniff. The slight rebound of the diaphragm when this test is being elicited appears to be due to the pendel-

luft effect. The paradoxical motion is a resultant of a combination of increased abdominal pressure acting on the paralyzed leaf from below, suction of the negative pressure of the intrapleural space above, and the increase of this negative pressure by the further shrinking of the partially compressed lung when the greater force of the other expanding lung draws air out of it, during forced inspiration.

SUMMARY

1. The value of the "sniff test" for the demonstration of successful paralysis of the hemidiaphragm following phrenic nerve interruption has been presented.

2. The basis for the test and its mechanics have been considered.

3. One hundred and nineteen phrenic nerve operations have been reviewed.

REFERENCES

1. Trudeau, F. B.: Phrenic Exeresis in the Treatment of Pulmonary Tuberculosis, J. A. M. A. **98**: 309, 1932.
2. Krogh, Fritz: Die temporäre Ausschaltung de N. phrenicus, Deutsche med. Wchnschr. **47**: 925, 1921.
3. Goetze, Otto: Temporäre Phreniksblockade, Zentralbl. f. Chir. **47**: 29, 1920.
4. Bakes, J.: Radikaloperation der Zwerchfellhernian mittels Kirschner's "Augelhackenschnittes" und temporärer Phrenicusblokade nach Perthes-Goetze, Zentralbl. f. Chir. **48**: 554, 1921.
5. Forsee, J. H.: Hemidiaphragmatic Paralysis in the Treatment of Pulmonary Tuberculosis, J. THORACIC SURG. **8**: 272, 1939.
6. Nehil, L. W., and Alexander, John: An Estimate of the Value of Phrenic Nerve Interruption for Phthisis on 654 Cases, J. THORACIC SURG. **2**: 549, 1933.
7. Stanbury, S. W.: Anatomical Changes in the Diaphragm Following Phrenicectomy, Am. Rev. Tuberc. **29**: 528, 1934.
8. Pickhardt, O. C., Stewart, W. H., and Thorburn, G.: Cinefluoroscopic Studies of Peculiar Breathing and Chest Motion, J. THORACIC SURG. **8**: 512, 1939.
9. Sauerbruch, F.: Chirurgie der Brustorgane, zweite Auflage, Berlin, 1920, Julius Springer.
10. Meyer, W.: Is Phrenicectomy or Exeresis Without Danger and as Simple as Assumed? Med. J. and Rec. **138**: 6, 1933.
11. Brauer, Rudolph: Ueber Pneumothorax, Marburg, N. G. Elwert'sche Verlagshandlung, 1906.
12. Moore, J. A.: Phrenicectomy in the Treatment of Pulmonary Disease; Analysis of 63 Cases, Arch. Surg. **20**: 175, 1930.
13. Sauerbruch, F.: Die Beeinflussung von Lugenerkrankungen durch künstlicher Lämung des Zwerchfelles (Phrenikotomie), München. med. Wchnschr. **60**: 625, 1913.
14. Walther, H. E.: Röentgenologische Untersuchungen über Wirkung der Phrenikotomie, Beitr. z. klin. Chir. **90**: 358, 1914.
15. Goetze, Otto: Die radicale Phrenikotomie als selbständiger therapeutischer Eingriff bei der chirurgischen Lungentuberkulose, Arch. f. klin. Chir. **121**: 244, 1922.
16. Alexander, John: Surgery of Pulmonary Tuberculosis, ed. 15, Philadelphia, 1925, Lea & Febiger, p. 175.
17. Miller, Felix P.: Avulsion of Phrenic Nerve, Texas State J. Med. **24**: 15, 1928.
18. Harrington, S. W.: Phrenicotomy as the Treatment of Diaphragmatic Hernia, and of Tumors of the Wall of the Chest, Arch. Surg. **18**: 561, 1929.
19. Werner, W. I.: Changes in the Respiratory Mechanism Following Phrenicectomy, J. A. M. A. **95**: 1162, 1930.
20. Lemon, W. S.: Physiologic Effect of Phrenic Neurectomy, Arch. Surg. **14**: 345, 1927.
21. Kiss, F., and Ballon, H. C.: Contribution to the Nerve Supply of the Diaphragm, Anat. Rec. **41**: 285, 1925.
22. Matson, Ray: Exeresis of Phrenic Nerve in the Treatment of Pulmonary Tuberculosis, Am. Rev. Tuberc. **22**: 1, 1930.

23. Hitzenberger, K.: Das Zwerchfell, Vienna, 1927, Julius Springer.
24. Bittdorf: Quoted by Matson, R., reference 22.
25. Brunner, A.: Die Chirurgische Behandlung der Lungentuberkulose, Leipzig, 1924, Johann Ambrosius Barth.
26. Cooper, A. T.: Phrenicoexairesis in Pulmonary Tuberculosis, Am. Rev. Tubere. **22**: 769, 1930.
27. Vadone, A.: Radiological Signs Permitting the Diagnosis of Paralysis of the Diaphragm Coincident With Pneumothorax, Am. Rev. Tubere. **20**: 736, 1929.
28. Vadone, A., and Udaondo, C. B.: The Pathogenic Mechanism of the Kienböck Phenomenon, Am. Rev. Tubere. **20**: 741, 1929.
29. Dowman, C. E.: Relief of Diaphragmatic Tic Following Encephalitis by Section of the Phrenic Nerve, J. A. M. A. **88**: 95, 1927.
30. Stuertz, L.: Künstliche Zwerchfellähmung bei schweren chronischen einseitigen Lungenkrankungen, Deutsche Med. Wchnschr. **37**: 2224, 1911.
31. Felix, W.: Anatomische, experimentelle und klinische Untersuchungen über den Phrenicus und über die Zwerchfellinnervation, Deutsche Ztschr. f. Chir. **171**: 283, 1922.
32. Kienböck, R.: Auf dem Röntgenschirm beobachtete Bewegungen in einem Pyopneumothorax, Wien. klin. Wchnschr. **22**: 538, 1898.
33. Brunn, H. B.: Surgical Principles Underlying One Stage Lobectomy, Arch. Surg. **18**: 490, 1929.

FLAP DRAINAGE OF RESIDUAL TUBERCULOUS CAVITIES

W. L. Rogers, M.D., Sidney J. Shipman, M.D., and A. C. Daniels, M.D. (by invitation), San Francisco, Calif.

DURING the last fifteen years a relatively large number of surgical procedures have been described as an aid in collapse therapy of apical lesions with large cavitation. Apical thoracoplasty involving the resection of a goodly portion of the upper rib cage (one to seven ribs) has been supplemented in many instances by more extensive procedures. Semb[1] and his followers advise an additional pneumolysis. Proust and Maurer have advised removal of transverse processes. Holman has recommended the additional removal of the lower portion of the scapula. Alexander[2] has recommended removal of full lengths of ribs and their cartilages as well as the transverse processes.

Various anterior procedures have been described as adjuncts. Head[3] has proposed an initial paraffin plombage in certain apparently difficult collapse problems; this to be followed, if necessary, by apical thoracoplasty. Butler, Skinner, Douglass, and Merkel[4] have recommended a myoplastic thoracoplasty with utilization of the pectoral muscles. Several supplemental procedures to thoracoplasty have also been described in an effort to collapse residual cavities. Matson has been using an extrapleural gauze pack in an endeavor to collapse residual cavities beneath thoracoplasty and of recent date has used it extensively as a primary procedure.

A much smaller group has recommended, from time to time, the advisability of draining persistent residual cavities either into an existing infected pleura or to the outside. Nissen[5] has reported a successful drainage of a large anterior apical cavity following an unsuccessful attempt at plombage. He utilized a muscle flap to fill the cavity space with favorable result. Lilienthal,[6-8] in 1927, 1929, and 1931, cited sporadic cases in two of which he had used the muscle flap similar to the case described by Nissen. He also described some other cases in which he had drained residual cavities, one into the pleura and three to the outside. In one case there was an apparent cure with spontaneous closure of the fistula. The others were apparently somewhat improved. He appeared to have some technical difficulty in keeping his wounds open. Coryllos and Ornstein[9] in discussing giant tuberculous cavities, in 1938, reported a series of twelve cases having residual cavities under thoracoplasty in which they had afforded open drainage and, at the same procedure, utilized a long pedunculated muscle flap to fill the cavity

Read before the Twenty-Fourth Annual Meeting of the American Association for Thoracic Surgery, Toronto, Canada, June 9, 10, and 11, 1941.

completely. They described a 50 per cent cure. However, their mortality rate was four in this group selected. In conclusion, they stated that while the muscle flap had given a 50 per cent negative sputum, it was a procedure still fraught with dangerous complications. Their results at attempting intracavitary cauterization of the communicating bronchus were not encouraging. Injection of material into cavities has not gained favor.

In reviewing statistical studies of immediate and late results of thoracoplasty, it would seem fair to conclude that we are all confronted with a small group of persistent residual cavities in spite of the type or character of mechanical collapse employed. O'Brien, Day, Chapman, and Tuttle,[10] reviewing 511 patients, stated that 447 of this group showed cavitation before operation. Following operation 387, or 87.02 per cent, had their cavities closed, and 58, or 12.98 per cent, remained open after operation. Dolly, Jones, and Paxton[11] in reviewing 1,636 cases found the sputum positive in 396 and described this to be due to (1) incomplete approximation of cavity walls, and (2) active bronchial or tracheal ulceration. In conclusion they cited the following reasons for cavity persistence:

1. Too long delay between stages
2. Posterior cavity protected by transverse processes not excised
3. Anterior cavity protected beneath the costal cartilages not removed
4. Insufficient length of ribs removed
5. Insufficient number of ribs removed

Have not most of these conclusions been based upon the concept that tuberculous cavities are merely vacant spaces hollowed by disease from the surrounding lung, neglecting the newer knowledge of cavity formation and persistence? Eloesser,[12, 13] in his classical work on bronchial stenosis, especially its relation to tuberculosis, and Coryllos and Ornstein, as well as Max Pinner and others, have pointed the way toward a dynamic concept of cavitation. This recognizes that the bronchial tree, as well as the surrounding lung, is not static but expansile and that the air passages themselves expand on inspiration and contract on expiration. In a diseased and tortuous condition such narrowed bronchi allow air to enter but make egress of air and morbid secretions extremely difficult. A one-way check valve is formed, blowing up, as it were, small rents in the lung structure until they become clinically recognizable cavities. These are spherical and have been termed tension, or blocked, cavities. It is now recognized that at times they offer stubborn resistance to the usual collapse measures.

Thus, it would seem that a modern concept of tuberculous cavitation would require that two factors be kept in mind: (1) The intrinsic or bronchial factor responsible for the one-way valve effect and consequent positive intracavitary pressures; and (2) the extrinsic or chest wall factor which sooner or later, by adhesions to the underlying pleura and

lung, may tend to keep the cavity from closing. It is the latter that has been attacked so vigorously and relatively successfully by various collapse measures, a few of which we have enumerated. This approach alone has, however, failed to close all cavities when used without regard to the intrinsic bronchial or positive pressure factor.

Experiments with needles and rubber tubes of various sizes in some of the tension cavities which have come under our observation have shown that simple exposure to atmospheric pressure will cause such a cavity to disappear at times in a very few minutes and that it will promptly reappear when the needle or tube is withdrawn. One of us (W. L. R.) has needled and withdrawn the air from such cavities immediately prior to thoracoplasty and watched them disappear. Following a standard thoracoplasty some have remained closed and others have reopened. Monaldi[14] has apparently cured cavities by the insertion of a catheter followed by suction. In our hands this procedure has generally failed, especially in large cavities (as Monaldi himself states), probably due to the fact that the intrinsic factor was satisfied but not the extrinsic. Certainly in our hands the Monaldi method has failed to close the type of cavity under discussion.

During the earlier stages of activity various areas in the upper portion of the lung field are apt to be affected as well as the area manifested by cavity formation. Doctor Vorwald has kindly consented to our use of the following lung sections demonstrating quite clearly this point (Fig. 1).

Therefore, bearing these factors in mind, it would seem logical and proper to start with thoracoplasty, more conservative procedures having failed. In all cases a routine upper first stage thoracoplasty was performed with the resection of all of the first and second and a liberal segment of the third rib. The electric coagulating current was used to destroy the periosteum on the posterior and lateral borders, as well as to control oozing and to seal lymphatics. Ten per cent formalin was used to touch the remaining anterior rib beds. This was followed by a second stage consisting of the removal of a liberal section of from two to four additional ribs. If three were taken, the lower portion of the scapula was generally removed to prevent distress from overriding on the seventh rib as well as possible to aid in the collapse obtained.

A goodly percentage of conversion will be effected by these relatively conservative procedures. Two-thirds of our group so treated are well and at work with negative or no sputum. No operative mortality was encountered in 100 consecutive cases. We feel that the remaining group, one-third of the cases in our series, should be carefully studied anew to determine, if possible, the probable source of the continued positive sputum. X-ray stereoscopic films, heavy density exposures, and planographs will aid in disclosing the diseased areas. Bronchoscopy may be of particular aid in determining the source and cause of the positive sputum.

The presence of tracheobronchial disease may also be recognized by this aid.

In the presence of active tracheobronchial disease, which would seem to be the source of the persistent positive sputum, further collapse therapy may seem undesirable. If the active disease would appear to be somewhat diffuse but still limited to the affected side, further resection downward or some revision modification would seem justifiable. However, if rather extensive study reveals an isolated, uncollapsed cavity underlying the resection as being the probable source of the continued positive sputum, we feel that the intrinsic or bronchial factor should be seriously considered and external decompression be performed rather than to resorting to further extensive revision procedures (Figs. 2 to 6).

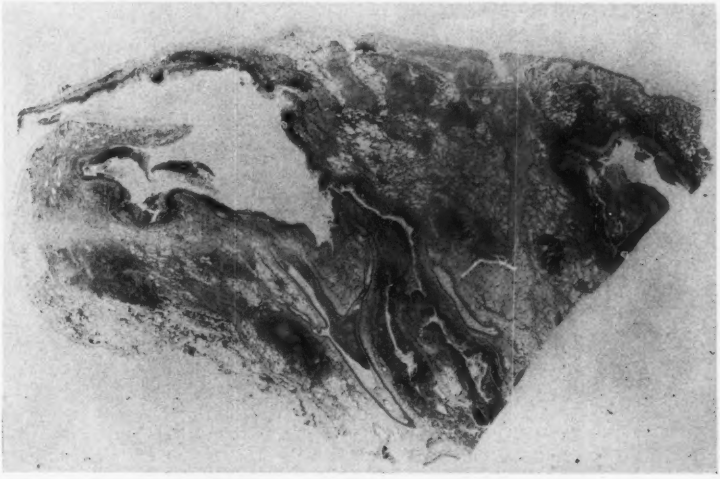

Fig. 1.—Large apical cavities with multiple smaller tuberculous foci in upper lung field (Vorwald).

To date, we have performed open cavity decompression in nine such cases with no mortality or serious complication. The five patients operated upon over a period of six months have all had sputum conversion and their wounds closed. The first case done with a slightly different technique, in 1936, required a subsequent closure of a bronchial fistula. The other four have had spontaneous closure of bronchial fistulas. All have remained well and healed to date. Of the remaining four patients operated upon within the last six months, all but one (the most recent) are afebrile. Two have had sputum and sinus conversion but still retain a small bronchial fistula with diminished cavity size in the bed of the wounds. The third has only a fleck of daily sputum, but may be classified as Gaffky I. His diminished cavity bed is still a Gaffky IV in contrast to a Gaffky VII at time of operation, and a small bronchial

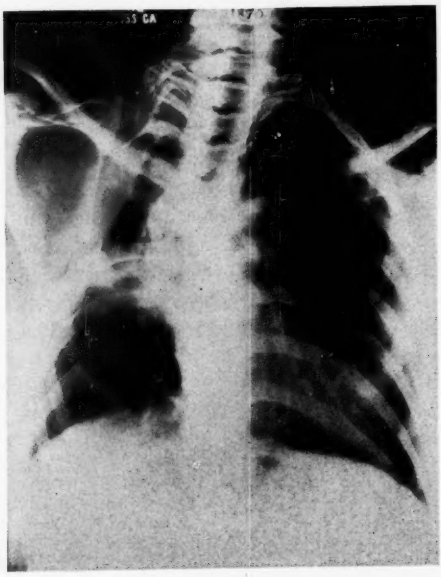

Fig. 2.—X-ray showing residual cavity.

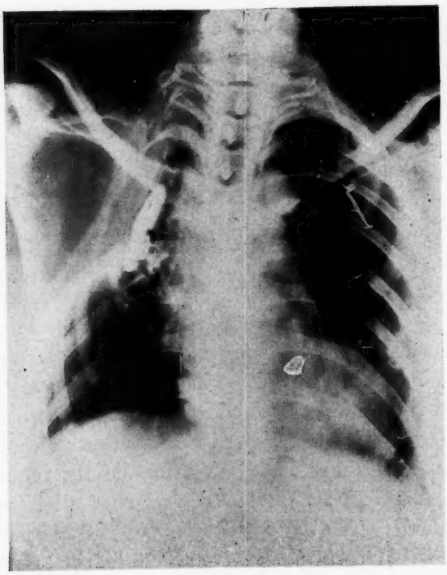

Fig. 3.—Cavity injected with lipiodol.

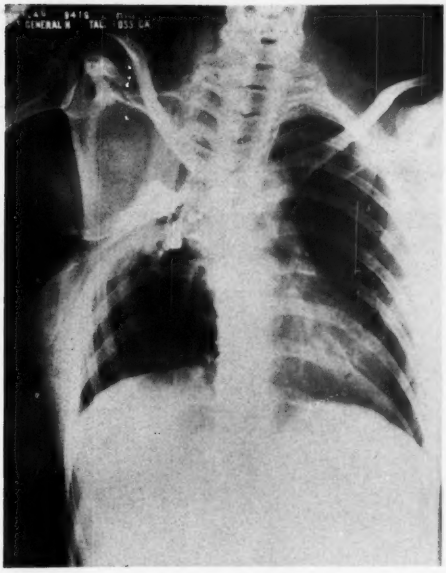

Fig. 4.—Lipiodol injected along skin flap directly into cavity.

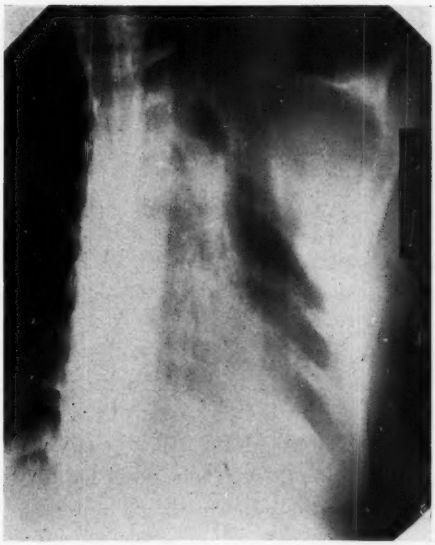

Fig. 5.—Planograph film revealing residual cavity under apical thoracoplasty.

communication is still present. The ninth case is too recent to discuss as to probable outcome. All cavities contained tubercle bacilli. One also contained pyogenic organisms, but in this case the secondary infection followed previous attempts at tubal drainage. Doctor Eloesser has used this type of drainage following essentially the same technique in two additional cases. In the first case operation was performed as a secondary procedure for a residual cavity beneath thoracoplasty with resulting

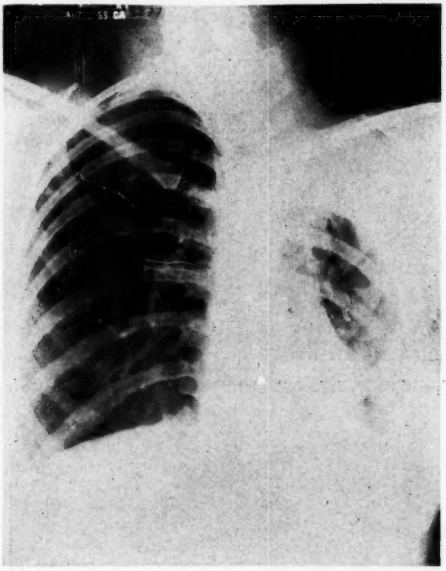

Fig. 6.—Cavity closed, sinus healed, sputum negative.

cure. In the second case it was utilized as a primary procedure. The indication in this instance was to aid, if possible, to diminish the amount of daily sputum by draining a rather large cavity in the middle lung field. The cavity was not considered the only source of the patient's sputum. General improvement followed. A sputum conversion was obviously not obtained.

Regarding technique, all operations have been carried out readily under local anesthesia. With the exception of the first case (operation 1936), the cavities were needled and readings recorded under various conditions (during normal respiration, following pressure against a closed glottis, after counting, after addition and also withdrawal of air). Two approaches have been utilized, a posterior or axillary, depending upon the location of the residual cavity (Figs. 7 and 8). A flap of skin has been utilized as described by Eloesser[15] in the treatment of chronic empyema. The apex of the flap is reflected away from the

wound, the underlying tissue severed with the aid of the actual cautery, any regenerated rib removed, and, if advisable, one intercostal muscle bundle with its nerve, artery, and vein also removed. A needle is then inserted into the cavity and readings are taken. With the needle in place, the intervening tissue is severed with the actual cautery and the cavity entered. A good-sized opening is made in the cavity wall, the cavity inspected, and smears and cultures are taken of its contents. The apex of

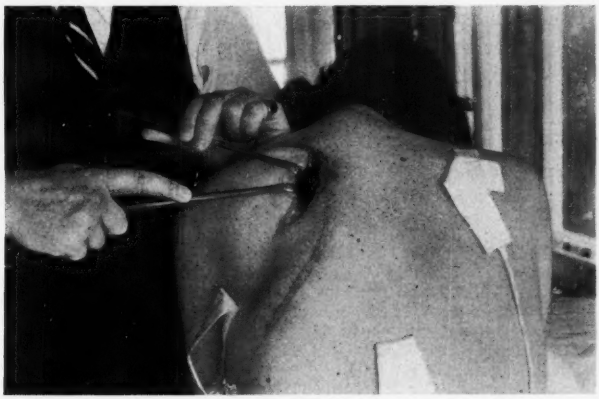

Fig. 7.—Posterior paravertebral approach.

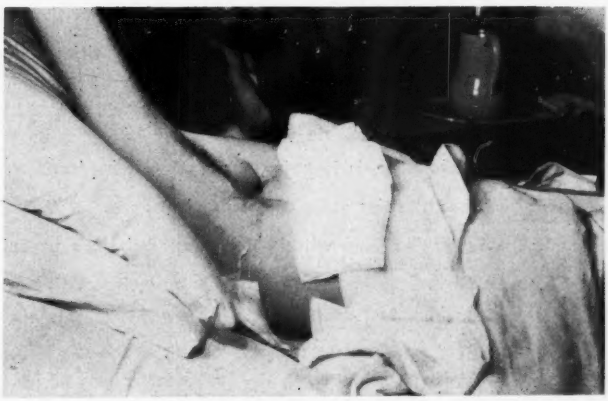

Fig. 8.—Anterior axillary approach.

the flap is then sutured to the cavity wall, or in most cases to the overlying thickened pleura with an interrupted mattress type of black silk suture. The cavity is then gently packed with plain or iodoform gauze and the wound dressed with vaseline strips.

In conclusion, we feel that a rational plan has been briefly discussed for the surgical treatment of apical pulmonary disease with cavitation,

bearing in mind the two important factors, namely, the intrinsic or bronchial factor and the chest wall factor, as being influential in cavity closure.

By following such a program, it is believed that a minimal amount of healthy lung will be sacrificed, and perhaps, at least in many cases, most extensive, somewhat mutilating, and at the same time hazardous procedures avoided. Specific indications have been outlined for open decompression, namely, a residual cavity remaining after a standard type of two-stage apical thoracoplasty in which the continued positive sputum would seem to be coming from the cavity itself and not from any adjacent foci. A satisfactory technique for this procedure has been described. A series of nine cases has been discussed with results obtained to date.

REFERENCES

1. Semb, C.: Thoracoplasty With Extrafascial Apicolysis, Acta chir. Scandinav. **76**: 1, 1935.
2. Alexander, John: The Collapse Therapy of Pulmonary Tuberculosis, Springfield, Ill., 1937, Charles C Thomas, Publisher.
3. Head, Jerome R.: Paraffin Pack and Thoracoplasty in the Closure of Large Apical Cavities, J. THORACIC SURG. **6**: 491, 1937.
4. Butler, E. F., Skinner, A. M., Douglass, R., and Merkel, C. G.: Myoplastic Thoracoplasty, J. THORACIC SURG. **8**: 93, 1938.
5. Nissen, R.: Gewebsplombierung einer durch die Brustwand perforierten Kaverne, Ztschr. f. Tuberk. **63**: 20, 1931.
6. Lilienthal, Howard: Open Treatment of Pulmonary Tuberculosis, Ann. Surg. **86**: 1927.
7. Lilienthal, Howard: Direct Drainage of Tuberculous Pulmonary Cavities, Arch. Surg. **19**: 1161, 1929.
8. Lilienthal, Howard: Operative Treatment of Phthisis, Am. J. Surg. **14**: 356, 1931.
9. Coryllos, P. N., and Ornstein, G. G.: Giant Tuberculous Cavities of the Lung, J. THORACIC SURG. **8**: 10, 1938.
10. O'Brien, E. J., Day, J. C., Chapman, P. T., and Tuttle, W. M.: Results in 511 Patients Subjected to Thoracoplasty, J. THORACIC SURG. **9**: 364, 1940.
11. Dolley, F., Jones, J. C., and Paxton, J. R.: Late Results of Thoracoplasty, Am. Rev. Tuberc. **39**: 145, 1939.
12. Eloesser, L.: Bronchial Stenosis in Pulmonary Tuberculosis, Am. Rev. Tuberc. **30**: 2, 1934.
13. Eloesser, L.: Blocked Cavities in Pulmonary Tuberculosis, J. Thoracic Surg. **7**: 1, 1937.
14. Monaldi, V.: L'Asperazione Endcavitaria Hella Cura della Caverne Tuberculosi del Polmone, Sett. med. **6**: 3, 1939.
15. Eloesser, L.: An Operation for Tuberculous Empyema, Surg., Gynec. & Obst. **60**: 1096-1097, 1935.

DISCUSSION

DR. EDGAR W. DAVIS, Washington, D. C.—In the latter part of last year, or perhaps the first of this year, an article came from Leipzig reporting four cases in which the Monaldi procedure had been used on residual cavities following thoracoplasty. In none of those four cases did the cavity eventually completely close and the sputum become negative.

According to the presentation of the papers today, we have approximately 70 or 75 per cent of good results following thoracoplasty. That still leaves a good number of unsatisfactory cases.

As has already been said, we are not satisfied with that; we should always strive to improve our results and encourage methods that offer hope in the unsatisfactory cases.

Several months ago, we had a young lady of about 27 years of age who had had a three-stage thoracoplasty in 1936. In 1938, she had a three-stage secondary thoracoplasty and a complementary anterior thoracoplasty with resection of the anterior segments, including the cartilages. As complete an anatomic collapse as was possible had been obtained. She still had a residual cavity 7 cm. in length and 4 cm. in width.

Since several people had advised against a lobectomy in some of those cases, it was felt that a surgical drainage of this cavity should be done, and if good results were not obtained, a lobectomy could be done later. Accordingly, the cavity was drained. We used the electrosurgical loop to remove sections of the wall of the cavity, and made histologic studies. The histologist was unable to distinguish any difference at all in the tissue removed from this cavity wall as compared with our routine, ordinary pyogenic abscess wall.

Unfortunately, about ten days or two weeks following surgical drainage, this girl developed scarlet fever. During the quarantine period her cavity was not packed widely. To avoid the complete closure of the opening into the cavity it was necessary to insert a rubber tube.

She had no unfavorable reaction, her temperature immediately became normal, the leucocytosis completely disappeared, her cough which had been very harassing completely disappeared, and her sputum, together with the secretions from the pulmonary cavity, immediately became negative.

It has now been about three or four months, and she is still doing exceptionally well, though her bronchial fistula is still open.

I have enjoyed the presentation of these papers and I feel that inasmuch as we have a certain percentage of residual cavities underlying thoracoplasty, there is certainly room for considerable improvement.

ACUTE PUTRID ABSCESS OF THE LUNG

V. Hyperacute Variety

Harold Neuhof, M.D., and Arthur S. W. Touroff, M.D.
New York, N. Y.

ALTHOUGH at the present time, considerable difference of opinion still exists concerning operative treatment for acute putrid pulmonary abscess, it is safe to say that thus far early surgical intervention has not been generally approved. The so-called conservative management of the disease is based upon two assumptions, namely, that cure occurs often without operation and that in the acute stage the presence of pneumonic infiltration renders surgical treatment unsafe. The first of these views rests upon a belief in the efficacy of such methods as postural drainage and bronchoscopy. The second is based upon the concept that abscess of the lung in the acute stage is not a localized process but is part of a more or less ill-defined and extensive area of inflammatory disease. Our argument in favor of operative treatment in the acute stage of pulmonary abscess has been set forth elsewhere[1, 2, 3] and need not be repeated here.

Regardless of the difference of opinion concerning the advisability of operative treatment in the acute stage of pulmonary abscess, no disagreement exists concerning the seriousness of the more severe cases of acute abscess and the lack of likelihood of cure of such cases under conservative therapy. Although relatively uncommon, these severe cases, which we have termed "hyperacute," are not rare in a large series of cases of pulmonary abscess. When treated by conservative methods, they tend to terminate fatally. Indeed, in all probability the mortality of the acute stage of abscess of the lung is low only because these cases are relatively rare. Thus, in view of the fact that spontaneous cure or cure by conservative methods cannot be anticipated, the necessity for some form of relief in these cases has been generally realized. As a result, they have been subjected to operation from time to time. Surgical treatment of cases of hyperacute pulmonary abscess has not been carried out routinely because, first, the patients often have been regarded as too ill for operative treatment; second, the lesion often has been considered too extensive; and third, the area of pulmonary destruction has been assumed to be only a part of a much more extensive

Received for publication, May 14, 1941.

ill-defined zone of "gangrene of the lung." The impression we have gained is that hyperacute abscess usually has been operated upon only because an error in diagnosis had been made and the lesion was considered to be a pyopneumothorax requiring prompt drainage. In fact, this was precisely the situation in our own first case, which shall now be reported briefly.

B. C., an adult male, was admitted to the Mount Sinai Hospital, Oct. 9, 1930, with a two weeks' history of cough and fever. One week after onset the sputum became intensely foul and copious and the patient became quite weak. At about the same time, glycosuria was discovered.

On admission, the patient was profoundly toxic and apathetic. Fever was of the septic type. The pulse rate varied between 88 and 124. X-ray examination revealed a large collection of fluid surmounted by air extending from the level of the third rib (anteriorly) to the apex. The patient was kept under medical observation and treatment for five days and then the question of surgical intervention came up, despite the desperate general condition of the patient, on the diagnosis of encapsulated pyopneumothorax due to a perforated acute putrid pulmonary abscess.

Fig. 1.—B. C., film of chest on admission to hospital; note large cavity with fluid level in right upper lobe, apparently an encapsulated pyopneumothorax. At operation the lesion was found to be a large unperforated pulmonary abscess.

At operation (Oct. 14, 1930), under local anesthesia, a segment of the third rib was excised and a globular cavity measuring about 10 cm. in all diameters was entered. The contents consisted of foul pus and pulmonary slough. Several bronchial fistulas were present and the cavity obviously was situated entirely within the substance of the lung and not within the pleura. After the lesion had been completely evacuated, the cavity was packed lightly with gauze.

The postoperative course was remarkably uneventful. Cough and expectoration ceased almost at once and the cavity rapidly became reduced in size. The patient was discharged entirely symptom-free in three weeks. The wound subsequently healed without incident and the patient remained free of all pulmonary manifestations until his death, four years later, from carcinoma of the stomach. At postmortem examination, the only significant findings referable to the former pulmonary disease were some local pleuritic adhesions over the upper lobe.

Fig. 2.—B. C., lateral film of chest; note that the lesion is situated in the anterior portion of the upper lobe.

Had the preoperative diagnosis in the foregoing case been correct, the patient would not have been operated upon because at that time (1930) such extensive lesions in patients in poor general condition were not regarded as amenable to operative relief. Only after a few similar experiences was it appreciated that cases of hyperacute abscess of the lung comprised an important group in which prompt operative treatment yielded remarkably good results.

Definition.—In "hyperacute" abscess the clinical course is severe and fulminating and the lesion usually is of large size. Obviously, hyperacute abscess is not defined sharply by this statement and certain cases

may be considered hyperacute by some and not by others. As a group, the cases are characterized by a short history, profound toxemia or apathy (with or without high fever), and a tendency to spread not only locally but also more remotely by the mechanism of bronchial spillover. The importance of segregating hyperacute cases lies in the necessity of regarding them as surgical emergencies irrespective of one's view in favor of or against early operation in less severe cases. Put in another way, hyperacute abscess can be properly regarded as one in which the likelihood of recovery under conservative therapy is not great, and the probability of a fatality is real, unless operative treatment is instituted.

Roentgen Features.—In a previous communication,[4] we stated that the roentgen features of ordinary cases of abscess of the lung are typical or characteristic only in the minority of cases. In the remainder, the diagnosis of pulmonary abscess cannot be made on the basis of roentgenograms alone. On the other hand, in almost all hyperacute cases that have come under our observation the films have been characteristic of abscess. Hence, it can be said that hyperacute abscess is characterized roentgenologically by the presence of a large area of excavation containing a fluid level. There are three additional roentgenographic features which are common in hyperacute abscess: (1) There is substantially more infiltration in the wall of the abscess than is seen ordinarily in acute abscess; (2) the abscess cavity may present an irregular or scalloped border due to the attachment of gangrenous pulmonary sloughs to the walls; and (3) the pleural reaction about the abscess often is both intense and extensive. Recognition of the last mentioned feature is of some importance because the diagnosis of an extensive, coexisting bronchopneumonia may erroneously be made if this fact is not borne in mind. Such an interpretation may lead to the assumption that the lesion is too extensive or too ill-defined for surgical intervention. Thus, in one of our cases such an error in roentgen interpretation led to some delay in instituting operative treatment. Fortunately, however, operation was performed and the patient recovered.

I. M., female, 43 years old, was admitted on March 21, 1939, with a history of fever, cough, and dyspnea of two days' duration. She was treated as a case of asthma and bronchopneumonia of the left lower lobe. The x-ray film demonstrated infiltration in the region of the apical portion of the lower lobe. Fever ranged to 105 F. and the patient expectorated small amounts of sputum. Sulfapyridine appeared to have no decisive influence on the clinical course. One week after admission the x-ray films revealed an area of rarefaction in the region of the previous pneumonic infiltration and a widespread invasion of the left lung or

pleura, or both. On the ninth day of hospital stay on the medical service, which was the eleventh day after the onset of the acute symptoms, the patient expectorated intensely foul sputum. The roentgenogram at that time revealed a fluid level in the area of rarefaction.

The patient's condition at this time was poor. There were cyanosis, dyspnea, profound prostration, and lethargy. She was confused and disoriented. The odor of the sputum was intensely and penetratingly foul, being that which is usually characterized as the odor of gangrene. Operation was proceeded with as a case of extreme emergency.

At operation, pronounced and edematous pleural adhesions were encountered through a paravertebral approach beneath the sixth rib, and a large gangrenous abscess of the lung was entered. The cavity was three inches or more in each diameter.

The change in the patient's condition after operation was striking. There was prompt cessation of expectoration of foul pus and rapid improvement in the general condition. The patient recovered quickly from a lethargic state which was so extreme that there was no response of any sort at the operation performed under local anesthesia. The sputum was never foul after operation, and cough and expectoration ceased three weeks after operation. The x-ray taken shortly after operation demonstrated the subsidence of the widespread reaction about the abscess. A subsequent film demonstrates the disappearance of all pulmonary infiltration.

An effort should be made to distinguish roentgenologically between a hyperacute abscess which is confined entirely to the substance of the lung and one which has perforated locally into the pleura. It may be stated, however, that such differentiation at times may be impossible, even at operation. The shell of lung overlying an unperforated abscess may be exceedingly thin or may not be recognizable as such at the time of operation because of the intensity of the local inflammatory reaction. In general, a perforated abscess is characterized roentgenologically by the presence of two overlapping fluid levels, one within the abscess and the other within the empyema. The latter is, of course, the more superficially situated and more broadly applied to the thoracic parietes, except in the unusual instances in which a pyopneumothorax is encapsulated within the interlobar fissure. Another point to be stressed is that separate pulmonary and pleural fluid levels may be distinguishable only if films are taken in several positions such as the lateral, oblique, or lateral recumbent.

Regardless of the question of whether or not a perforation exists, the roentgenologic demonstration of the precise site of contact between the lesion and the thoracic parietes is imperative in all cases of hyperacute abscess. Theoretically, one might assume the existence of a wide area of contact and, hence, of widespread adhesions in all cases of hyperacute abscess because, as already stated, such lesions are of substantial size. That this assumption may not be correct, was proved by one of our earliest experiences.

J. P., a male, 50 years of age, admitted Oct. 11, 1932, who was profoundly toxic, came under observation with a huge lesion which appeared to occupy most of the upper half of the right lung. At operation, performed at a site chosen rather at random, over the anterior chest, pleural adhesions were not encountered. The pleural cavity, fortunately, was not entered and operation was discontinued. Further roentgenologic studies then demonstrated that the sole area of contact of the abscess with the parietal pleura was situated posteriorly. At the second operation, performed a few days after the first, the abscess was entered in one stage through walling-off pleural adhesions. Had the free pleural cavity been traversed at the first operation, making necessary a second stage procedure at that obviously unfavorable site, recovery might not (probably would not) have ensued.

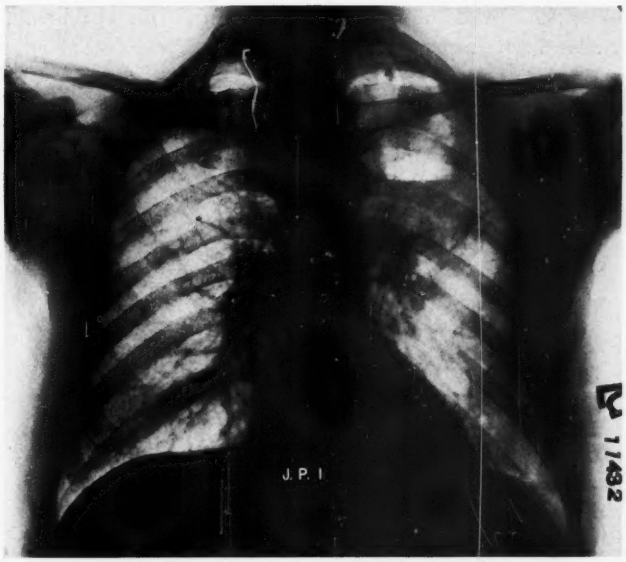

Fig. 3.—J. P., film of chest on admission; note large cavity with fluid level in left upper lobe. The lesion is of such large size that it apparently can be approached surgically either from the anterior or posterior aspect of the thorax.

Precise roentgenographic localization of the abscess (with its corollary of a safe one-stage operation) appears to be even more imperative in cases of hyperacute abscess than in cases of less severe forms of acute pulmonary abscess because of the necessity of eliminating promptly the source of infection and of cough and purulent expectoration.

Indications for Operation.—Assuming rather general agreement as to the advisability of early operation in cases of hyperacute abscess, the only question to be discussed is that of contraindications to operation. In view of the fact that we have repeatedly seen quite unexpected recoveries after drainage of enormous abscesses in gravely ill patients (others undoubtedly have had similar experiences), there seems to be

no contraindication to operation other than an actually moribund patient. The hyperacute variety of pulmonary abscess is the only one in which an appreciable surgical mortality should exist and that mortality should be faced unhesitatingly, for in all likelihood the patients will die unless adequately relieved by operation.

Operative Treatment.—The imperative necessity of a one-stage operation, which has already been stressed, should be the keystone of surgical treatment. The prerequisite is precise roentgenographic localization of the lesion. Little time is required for this essential preoperative step and it therefore must not be omitted even in cases in which operation is urgent. The details of technique of operation for acute pulmonary abscess have been fully set forth elsewhere.[5] They consist essentially

Fig. 4.—J. P., film taken after unsuccessful attempt at operation through an anterior approach; note that the lesion lies far posteriorly.

in the exposure and entry of the abscess through existing visceroparietal adhesions at the site of contact between the lesion and the chest wall. The abscess is unroofed by excising, within the limits of adhesions, the thin avascular shell of overlying lung. After wide entry of the abscess, evacuation of its contents, and exploration of recesses (all under full visualization), the operation is completed by packing the cavity

lightly with gauze. In cases of perforated abscess of the variety under consideration, the pulmonary abscess as well as the pyopneumothorax must be unroofed.

It is probable that the outcome is more directly related to the operative procedure in hyperacute abscess than in ordinary cases of acute abscess. The reason lies in the fact that the patient is more critically ill and hence less likely to recover if the operative procedure falls short in any important particular. The rather natural tendency toward the performance of a more or less incomplete operation, because of the patient's condition, therefore should be avoided. Accordingly, emphasis should be placed on some of the operative steps of the single-stage procedure. Whereas local anesthesia may be regarded as elective in ordinary cases of pulmonary abscess, it is imperative in hyperacute abscess. Care should be taken to excise segments of rib only to the extent required for unroofing the abscess, in order to avoid the possibility of opening the free pleura, either by an inadvertent incision or by coughing or straining efforts on the part of the patient. On the other hand, the unroofing of the abscess should not be meager, because liberal unroofing provides the best means for prompt elimination of the anaerobic infection and, thus, of the toxic or septic state. Since the overlying lid of lung is apt to be quite necrotic in cases of hyperacute abscess, it should be excised with great precision. Any recesses of the main abscess cavity should be explored and adequately laid open in order to avoid persistence of infection. Gangrenous sloughs should not be disturbed when adherent. In short, every effort should be made to provide free evacuation of infection with minimal trauma.

Results of Operation.—Our series of operations for acute putrid pulmonary abscess consists now of 122 cases, with 4 deaths, a mortality of 3.27 per cent. When the hyperacute cases are split off, the results are as follows: 38 hyperacute cases, 3 deaths, mortality 7.9 per cent; 84 other cases, 1 death, mortality 1.19 per cent.* Thus, it can be stated that the only group of cases of acute putrid pulmonary abscess in which a substantial mortality must be faced at the present time is the hyperacute variety and it is precisely this group in which a much higher mortality undoubtedly exists if operative relief is not given. Hence, the indication for operation exists in all cases of hyperacute putrid pulmonary abscess regardless of the views which may be held concerning less severe or fulminating cases. With precise localization and a one-stage operation through visceroparietal pleural adhesions, recovery often will take place when scarcely anticipated at the time of operation.

*Since the submission of this paper there have been thirty-two additional operations for acute abscess, without additional deaths. Thus, the present mortality of operation is 2.6 per cent. Of the additional operations, three were for cases of the hyperacute variety, the mortality of this group also being lowered thereby.

REFERENCES

1. Neuhof, H., and Touroff, A. S. W.: Acute Putrid Abscess of the Lung; II. An Analysis of Forty-Five Consecutive Operative Cases, Surg. Gynec. & Obst. **66**: 836-857, 1938.
2. Neuhof, H., and Touroff, A. S. W.: Acute Putrid Abscess of the Lung; A Surgical Disease, New York State J. Med. **40**: 1940.
3. Neuhof, H., and Touroff, A. S. W.: Acute Putrid Abscess of the Lung; IV. Surgical Treatment and Results in Eighty-Six Consecutive Cases, J. THORACIC SURG. **9**: 439, 1940.
4. Neuhof, H., and Touroff, A. S. W.: Acute Putrid Abscess of the Lung; III. Roentgenographic Features, Surg. Gynec. & Obst. **68**: 687-698, 1939.
5. Neuhof, H., and Touroff, A. S. W.: Acute Putrid Abscess of the Lung; Principles of Operative Treatment, Surg., Gynec., & Obst. **63**: 353-368, 1936.

In Memoriam

Frederick Taylor Lord
1875-1941

Dr. Frederick Taylor Lord, president of the American Association for Thoracic Surgery in 1932, died in Boston, Nov. 4, 1941. He achieved recognition as a scientist and scholar despite the distractions of a busy consultation practice that called him far and wide throughout the New England States. A sustained and rigid insistence on factual observation ever characterized his efforts in the laboratory, at the bedside, and in the lecture room.

He was born in Bangor, Maine, Jan. 16, 1875. Graduating from the Harvard Medical School in 1900, Dr. Lord acquired his training and experience in clinical medicine at the Massachusetts General Hospital, first as Medical House Officer, and subsequently as a member of the staff of that institution. From 1912 to 1935, he was visiting physician and after 1935, a member of the board of consultation. He organized and for many years directed the thoracic clinic. A member of the faculty of the Harvard Medical School from 1905, he held the rank of clinical professor of medicine from 1930.

Although broadly experienced and competent in internal medicine, diseases of the respiratory tract became his special interest after early work (1902) on the influenza bacillus. Pneumonia and lung abscess were major subjects of study and publication in later years. Investigation of the use of antipneumococcus serum and an interest in the control of tuberculosis led to active participation in public health programs in these fields.

In 1915, appeared his book, *Diseases of the Bronchi, Lungs and Pleura*, revised in 1925. This monograph is based on a thorough knowledge of the literature and an analysis of carefully recorded original clinical observations.

For services in devasted areas of Monastir in 1917, Dr. Lord was awarded the Second Order of St. Sava and the Red Cross (both of Serbia.)

In the examination of a patient, Dr. Lord showed the same meticulous attention to detail that characterized his teaching and research. He was not one to indulge in brilliant (when correct) intuitive diagnosis, but painstakingly assembled a series of facts by a personally taken history and complete physical examination. These facts were translated into an appraisal of the situation advanced as an hypothesis rather than a proved conclusion. To him a diagnosis was an intellectual decision

determined by preponderance of evidence and always subject to revision in the light of new evidence. It was never a subjective "hunch" defended by appeal to inarticulate "experience."

A classicist in the art of auscultation and percussion, increasing dependence on the x-ray examination never detracted from his interest in and skillful interpretation of the findings of the stethoscope.

Dr. Lord's experience with thoracic surgery started when he was a consultant in operating rooms where it was necessary for him to tell the surgeon what rib to resect in order to drain an empyema or lung abscess. He was cautious and conservative during the fumbling developmental years of this branch of surgery. He never "blew hot" with the enthusiasms of the moment, nor "cold" when unforeseen disaster might well have halted progress. On the other hand, his habits of critical and objective appraisal of results led him to grow apace with the subject. No one derived more intense personal satisfaction from the achievements of the thoracic surgeons during the past decade than Dr. Lord.

Always generous in deference to observations (but not casual opinions) of students and colleagues, ever humble in trusting his own ability to interpret the phenomena of disease, Dr. Lord will be remembered as a true scholar, an inspiring teacher, and a gentle, wise physician.

Edward D. Churchill.

Dr. Lord, although not a surgeon himself, recognized fully the greater potentialities of a surgical over a nonsurgical treatment of many thoracic ailments, provided that a surgical attack could be made reasonably safe. For that reason he joined the American Association for Thoracic Surgery in its early years and he was a regular and enthusiastic attendant at the meetings. No one was more pleased than he at the steady progress of this special field. From the time of the founding of THE JOURNAL OF THORACIC SURGERY until his death last year, he was a member of the Advisory Editorial Board.

Evarts A. Graham, editor.